

Diagnostic Performance of Serum IL-10 and IL-17 and Their Association with Disease Severity in Entamoeba Histolytica Infection

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Abstract: Background: Entamoeba histolytica is the etiologic agent of amoebiasis, a prevalent parasitic disease and a major public health problem worldwide, especially in developing countries. The severity of the disease depends on the parasite's virulence and the host's immune response. Interleukin-10 (IL-10) is a well-known immunoregulatory cytokine. IL-17 is a well-known pro-inflammatory cytokine. Therefore, their concurrent elevation during E. histolytica could be diagnostically useful and have important immunological implications. Methods: A case-control study was conducted in 45 laboratory-confirmed patients diagnosed with E. histolytica infection and 45 healthy controls. Patients were classified into mild (n = 9), moderate (n = 10), and severe (n = 26) groups based on disease severity. The serum levels of IL-10 and IL-17 were measured by a quantitative sandwich enzyme-linked immunosorbent assay (ELISA) kit. Differences between severity groups were analyzed using a one-way analysis of variance (ANOVA) followed by Duncan's multiple range test. The diagnostic performance of both cytokines was assessed by receiver operating characteristic (ROC) curve analysis. Statistical significance was set at $P < 0.05$. Results: Serum levels of IL-10 and IL-17 were markedly elevated in patients with E. histolytica infections compared to healthy controls. The cytokine levels gradually increased with the severity of the disease, and the highest levels were observed in patients with more severe clinical symptoms ($P < 0.001$). ROC curve analysis revealed excellent diagnostic performance for both cytokines: IL-10 showed remarkable discriminative ability (AUC = 0.995, sensitivity 95.5%, specificity 95.5%), and IL-17 showed excellent discriminative ability (AUC = 0.935, sensitivity 93.2%, specificity 82.6%). Conclusion: Serum levels of IL-10 and IL-17 were significantly associated with disease severity and showed an excellent diagnostic performance in patients with E. histolytica infection. The simultaneous increase of these functionally distinct cytokines may reflect the simultaneous activation of inflammatory and immunoregulatory immune responses during acute amoebic infection. These findings indicate the possibility to use the two cytokines as additional immunological biomarkers for evaluation of disease severity and diagnostic assessment of amoebiasis. Further investigation into the biological significance of the co-elevated levels of both cytokines is required.	Research Paper *Corresponding Author: Ayat Hassan Ali Department of Biology, College of Science, University of Al-Qadisiyah How to cite this paper: Ayat Hassan Ali & Esraa Fadhil Wadhah; "Diagnostic Performance of Serum IL-10 and IL-17 and Their Association with Disease Severity in Entamoeba Histolytica Infection" Middle East Res J. Microbiol Biotechnol., 2026 Jul-Aug 6(3): 173-178. Article History: Submit: 21.06.2026 Accepted: 23.07.2026 Published: 04.08.2026
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INTRODUCTION

Entamoeba histolytica remains one of the most important protozoan causes of intestinal infection worldwide and continues to pose a substantial burden in low- and middle-income countries where sanitation, access to clean water, and diagnostic resources may be limited (Cooney *et al.*, 2025). Although many infections remain asymptomatic, a clinically significant proportion progress to intestinal amebiasis, dysentery, or extraintestinal complications, particularly amoebic liver

abscess, making the parasite highly relevant in both gastroenterology and infectious disease practice (Cooney *et al.*, 2025). Recent reviews have also emphasized that amebiasis may mimic inflammatory bowel disease, creating a risk of misdiagnosis and inappropriate immunosuppressive treatment, which can worsen disease outcomes (Cooney *et al.*, 2025).

The pathogenesis of *E. histolytica* is multifactorial and reflects a dynamic interaction between parasite virulence, host mucosal defense, immune

regulation, and the intestinal microbial ecosystem (Cruz-Baquero *et al.*, 2023; Uribe-Querol & Rosales, 2020). Trophozoites colonize the intestinal lumen and may adhere to colonic mucus and epithelial cells through the Gal/GalNAc lectin, followed by secretion of cysteine proteases, pore-forming peptides, and other virulence factors that promote epithelial disruption, mucus degradation, and tissue invasion (Uribe-Querol & Rosales, 2020; Begum *et al.*, 2015). More recent work has further highlighted the role of extracellular vesicles and parasite-derived immunomodulatory signals in shaping macrophage responses and altering the inflammatory microenvironment during infection (2024; Chowdhury *et al.*, Verma *et al.*, 2024).

Clinical outcome is strongly influenced by the host immune response. Protective immunity against *E. histolytica* involves the coordinated action of epithelial barriers, mucins, antimicrobial peptides, neutrophils, macrophages, and adaptive immune mediators (Moonah *et al.*, 2021; Nakada-Tsukui & Nozaki, 2016). However, the same inflammatory pathways that help restrict parasite invasion may also contribute to tissue damage when dysregulated. This duality makes cytokines particularly informative indicators for understanding disease severity and host-pathogen interactions (Uribe-Querol & Rosales, 2020; Nakada-Tsukui & Nozaki, 2016).

Among the cytokines implicated in intestinal amebiasis, interleukin-10 (IL-10) is of particular interest because of its major anti-inflammatory and mucosal homeostatic functions. IL-10 helps preserve epithelial integrity, suppress excessive inflammatory signaling, and regulate immune-mediated tissue injury at mucosal surfaces (Nakada-Tsukui & Nozaki, 2016; Uribe-Querol & Rosales, 2020). Experimental and translational evidence indicates that IL-10 is important for limiting destructive intestinal inflammation during amoebic infection, yet elevated IL-10 may also reflect a compensatory response to more severe tissue invasion and dysenteric disease (Nakada-Tsukui & Nozaki, 2016; Mortimer & Chadee, 2010). This apparent dual role makes IL-10 a biologically plausible marker of both host protection and disease severity.

Interleukin-17 (IL-17) is another cytokine of growing relevance in amebiasis research because it is closely linked to mucosal inflammation, neutrophil recruitment, antimicrobial peptide induction, and epithelial defense (Moonah *et al.*, 2021; Uribe-Querol & Rosales, 2020). IL-17-mediated pathways may contribute to early protective responses against parasite invasion, particularly through reinforcement of innate immunity at the intestinal surface (Nakada-Tsukui & Nozaki, 2016). At the same time, higher IL-17 concentrations may also indicate intensified inflammatory activity in symptomatic or severe disease. Recent clinical work has shown increased IL-17 levels in

infected children, further supporting the relevance of this cytokine in the host response to *E. histolytica* infection (Aljanabi *et al.*, 2025).

In parallel with immunological advances, diagnostic approaches to amebiasis have improved considerably. Classical stool microscopy remains widely used in endemic settings, but it has limited sensitivity and cannot reliably distinguish *E. histolytica* from morphologically similar nonpathogenic species such as *E. dispar* (Fotedar *et al.*, 2007; Cooney *et al.*, 2025). Recent reviews have highlighted the increasing value of antigen-based assays, molecular methods, and species-specific diagnostics for identifying invasive disease more accurately (Cruz-Baquero *et al.*, 2023; Cooney *et al.*, 2025). Nevertheless, laboratory capacity remains uneven across many healthcare settings, and there is continued interest in adjunctive biomarkers that may reflect both disease activity and discriminatory ability between infected individuals and controls.

Despite these advances, clinically relevant gaps remain. Many studies have addressed the general immunobiology of amebiasis, but fewer have focused specifically on the combined behavior of IL-10 and IL-17 in relation to symptom severity and diagnostic performance in well-defined patient groups (Nakada-Tsukui & Nozaki, 2016; Aljanabi *et al.*, 2025). Given the complementary biological roles of IL-10 as a regulatory cytokine and IL-17 as a pro-inflammatory mediator, evaluating both markers may provide a more integrated picture of the immune landscape of intestinal amebiasis. Furthermore, if these cytokines differ systematically between mild, moderate, and severe cases, they may have value not only in understanding pathogenesis but also in supporting clinical stratification.

Accordingly, the present study was designed to evaluate serum IL-10 and IL-17 levels in patients with *Entamoeba histolytica* infection, to examine their association with disease severity, and to assess their diagnostic performance. By linking cytokine measurements with clinical severity categories, this work aims to contribute to the growing effort to identify immunological markers that are both biologically meaningful and clinically useful in amebiasis (Cooney *et al.*, 2025; Cruz-Baquero *et al.*, 2023).

MATERIALS AND METHODS

Study Design and Participants

This case-control study was carried out from 15 November 2025 to 25 April 2026, after recruiting a total of (90) participants who were recruited from Al-Diwaniyah Teaching Hospital and the Maternity and Children's Hospital, Al-Diwaniyah, Iraq. Participants were aged 1–56+ years. The study population was segregated into patients with laboratory-confirmed *Entamoeba histolytica* infection (n = 45) and a

comparison group ($n = 45$) comprising apparently healthy individuals admitted to the hospital. At the time of enrollment, control participants were confirmed not to have parasitic infections and reported no pre-existing chronic or inflammatory diseases.

Demographic and Clinical Data Collection

Demographic and clinical data were obtained for all participants using a structured questionnaire. The collected variables included age, sex, place of residence (urban or rural), infection status, dysentery status, treatment status, and clinical severity of infection. These variables were used to characterize the study population and to assess their association with serum cytokine levels and disease severity.

Clinical Severity Classification

Infected individuals were classified into three clinical groups according to severity of disease: Mild cases were defined as patients present with mild intestinal symptom. The moderate cases were those with suggestive amebiasis manifestations: diarrhea from 3 to 9 times per day associated with abdominal cramps. Severe case definition in our setting was: acute manifestations include dysentery with bloody or mucoid diarrhea, high fever and visible evidence of dehydration. We used these grade of severity to investigate the association between cytokine levels and clinical outcome in *E. histolytica* infection.

Blood Collection and Serum Processing

Venous blood (five milliliters) was collected from all subjects by sterile standard venous aspiration. Blood collected in three milliliter gel tubes was set at room temperature to clot. After that, samples were centrifuged to 4000 rpm in 10 min and the serum was separated and put into sterile Eppendorf tubes. Serum samples were stored at -20°C until analysed.

Measurement of Serum IL-10 and IL-17

The concentration of interleukin-10 (IL-10) and interleukin-17 (IL-17) in serum were determined using commercially available quantitative sandwich enzyme-linked immunosorbent assay (ELISA) kits, according to the manufacturers' instructions. For both assays, the microtiter plates were coated with monoclonal antibodies that bind to the target cytokine. Serum samples/standard solutions were added to designated wells, followed by biotinylated detection antibodies and streptavidin-horseradish peroxidase conjugate, forming antigen-antibody complexes. After incubation, washing steps are performed before accepting chromogenic and color developer substrate for color development the reactions were then stopped with stop solution. The absorbance was determined at 450 nm using a microplate reader, and cytokine concentrations were measured according to the standard calibration curves.

IL-10 ELISA Procedure

Before use, all reagents and standards were equilibrated to room temperature with serum samples for IL-10 quantification. From 1600 pg/mL of original standard concentration, the stem line curve was prepared by serial two-fold dilution to afford working concentrations for a range from 800, 400, 200, 100 and finally down to zero with the diluent for two further dilutions alone serve as the zero standard. Diluting 20 mL of the concentrated wash solution with 480 mL of deionized water gave a final volume of 500 mL, which was used to prepare a $1\times$ wash buffer. In this assay, the standard solution in each concentration was added to the standards wells: 50 μL were diluted and used for the dilution of 10–100 pmol/50 μL . Forty microliters (μL) of serum sample was added along with 10 μL anti-IL-10 antibody and 50 μL streptavidin-HRP conjugate to the sample wells. The plate was then incubated at 37°C for 60 min, washed five times with $1\times$ wash buffer, and subsequently incubated with an equal amount of Chromogen Solutions A (50 μL) and B (50 μL) in the dark at 37°C for 10 min. The reaction was terminated with 50 μL of stop solution before absorbance was measured at 450 nm after 10 min.

IL-17 ELISA Procedure

Prior to running the assay, all reagents and microplate strips were brought to room temperature for IL-17 quantification. Serial two-fold dilutions from the original standard concentration of 640 ng/L were prepared to create concentrations of 320, 160, 80, 40 and 20 ng/L for preparing the standards while the zero standard was prepared using the standard diluent. To prepare a $1\times$ wash solution, dilute 20 mL of wash buffer ($25\times$ concentrated) with 480 mL of distilled or deionized water. 50 μL of each standard was added to the respective wells assigned for the assay. Twenty microliters of serum were added to the sample wells, along with 10 μL of anti-IL-17 antibody and 50 μL of streptavidin-HRP conjugate. After incubation, the microplate was first washed with 100 μL of $1\times$ wash buffer five times per well, and then incubated at 37°C for another 10 min in the dark with each Chromogen Solutions A and B (50 μL). The reaction was stopped by the addition of 50 μL stop solution, and optical density was read within 10 min at a wavelength of 450 nm with a microplate reader pre-calibrated with the same stop solution.

Statistical Analysis

Statistical analysis was performed using SPSS software version 26.0. Data were expressed as mean $\pm$ standard deviation (SD). Differences in serum IL-10 and IL-17 concentrations among the disease severity groups were analyzed using one-way analysis of variance (ANOVA). Receiver operating characteristic (ROC) curve analysis was performed to evaluate the diagnostic performance of IL-10 and IL-17, including calculation of the area under the curve (AUC), sensitivity, specificity,

and optimal cutoff values. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Serum IL-10 and IL-17 Levels According to Disease Severity

The serum concentrations of IL-10 and IL-17 were assessed in patients with *Entamoeba histolytica* infection according to disease severity. A progressive elevation in both cytokines was observed with increasing

severity of infection. IL-17 levels increased from 151.196 ± 56.192 in mild cases to 276.00 ± 170.623 in moderate cases and 457.384 ± 123.923 in severe cases, with a highly significant difference among the groups ($p < 0.001$). Likewise, IL-10 levels showed a marked stepwise increase, with mean values of 247.564 ± 45.827 , 515.666 ± 104.431 , and 777.968 ± 211.056 in mild, moderate, and severe cases, respectively, and the differences were highly significant ($p < 0.001$). Post hoc group labeling indicated significant differences among all severity categories for both markers.

Table 1: Serum concentrations of IL-10 and IL-17 according to severity of infection

Parameters	Severity of infection Mean $\pm$ Sd.			P value
	Mild (n=9)	Moderate (n=10)	Sever (n=26)	
IL-17	151.196 ± 56.192^c	276.00 ± 170.623^b	457.384 ± 123.923^a	.000*
IL-10	247.564 ± 45.827^c	515.666 ± 104.431^b	777.968 ± 211.056^a	.000*

*Significant difference at the 0.05 level by One-way ANOVA test. NS: N-significant.
Different letters referred to significant differences between groups by Dunken test

Different superscript letters indicate significant differences between groups according to Duncan's multiple range test, and significance was determined by one-way ANOVA at the 0.05 level.

Diagnostic Performance of IL-10 and IL-17

Receiver operating characteristic (ROC) curve analysis was performed to evaluate the diagnostic accuracy of IL-10 and IL-17 in distinguishing infected patients from controls. Both biomarkers demonstrated statistically significant diagnostic value. However, IL-10

showed superior overall diagnostic performance compared with IL-17.

IL-17 yielded an AUC of 0.935, with a sensitivity of 93.2% and a specificity of 82.6% at a cutoff value of 97.1154. In contrast, IL-10 demonstrated an AUC of 0.995, with sensitivity and specificity of 95.5% each at a cutoff value of 136.917. These findings indicate that IL-10 had the highest discriminatory power and may represent the more reliable diagnostic biomarker in the studied population.

Table 2: ROC curve analysis of IL-10 and IL-17 in patients with *Entamoeba histolytica* infection

Markers	AUC	Sensitivity	Specificity	Cut-off	P-value
IL-17	0.935	0.932	0.826	97.1154	.000*
IL-10	0.995	0.955	0.955	136.917	.000*

AUC: area under the curve; *Significant difference at the 0.05 level by ROC analysis

AUC denotes area under the curve, and statistical significance was established by ROC curve analysis at a 0.05 level.

Overall Findings

Overall, serum IL-10 and IL-17 levels increased progressively with disease severity, and both demonstrated excellent diagnostic performance in patients with *E. histolytica* infection. These findings indicate that both cytokines are closely associated with disease severity and may represent useful adjunct immunological biomarkers for the laboratory evaluation of amoebiasis.

DISCUSSION

In addition, this study showed that both serum IL-10 and IL-17 levels were increased according to the clinical severity of *E. histolytica* infection with a tendency for greater values among patients presenting more severe disease. Furthermore, both cytokines were significantly predictive regarding diagnosis but only IL-10 discriminated better than IL-17 (higher AUC and

respectively sensitivity as well as specificity). The data presented here also support the hypothesis that host cytokine responses correlate with tissue invasion and clinical disease, not just amebic infection. The orderly rise in IL-10 with mild, moderate and severe cases is biologically plausible and may reflect an enhanced anti-inflammatory response to increased mucosal injury. IL-10 is an indispensable immunoregulatory cytokine that needs to maintain the integrity of intestinal barriers, restrict excessive inflammatory responses and inhibit NF- κ B-inducing epithelial activation in contributing MUC2 production and IgA-related mucosal protection. Results evidence supports that innate resistance to amebic colitis requires IL-10, with reduced susceptibility due to IL-10 dependency, and increased invasiveness in the absence of IL-10. Therefore, the high levels of IL-10 was detected here in cases that were more severely ill may have been a consequence of the increasing

penetrance and dysenteric disease rather than being purely indicative of protective immunity. This interpretation agrees with earlier studies showing raised levels of IL-10 during dysenteric and invasive amebiasis but not in asymptomatic carriers of infection [11]. At the same time, the very high levels of IL-10 observed during severe infection may indicate excess immunoregulation. The phenomenon would be accompanied by the consequent of offense and might have a redefining substitutive role in unintentional parasite efficiency inhibitor for select hosts. *E. histolytica* employs different strategies for immune evasion, including modulation of the macrophage and epithelial cell signaling, induction of anti-inflammatory pathways mediated by prostaglandins (e.g., cyclooxygenase-2), degradation of immune mediators (for example, TNF- α mitogen-activated protein kinase) and inhibition of a sufficiently effective pro-inflammatory response required for parasite clearance. A host-parasite interaction may create a setting in which IL-10 is produced through a regulatory loop that maintains homeostasis to limit tissue destruction while coupling this immune event to ongoing invasion. Based, IL-10 in amebiasis is critical for mucosal homeostasis but may also play a protective and pathological role depending on concentration: Low levels being essential for maintaining an appropriate immune response while high levels are hallmark of more severe/invasive disease. The correlation between IL-17 and disease severity in this study is also consistent with the pleiotropic role of IL-17 in mucosal defense and intestinal inflammation. IL-17 promotion leads to neutrophils recruitment, antimicrobial peptides induction, mucin secretion enhancement and epithelial immune function reinforcement. IL-17 mediated responses can participate in protective immunity to *E. histolytica* as suggested by experimental studies, mediated through stimulation of mucosal defenses and coordination of innate immune cell trafficking. Thus, in our study the increase of IL-17 from mild to severe cases may therefore represent a stronger activation of inflammatory and barrier-defense pathways due to greater parasite invasion. Still, since severe amebiasis is associated with profound epithelial disruption, bloody diarrhea and inflammatory injury, high IL-17 may also reflect an ongoing inflammatory burden rather than wholly effective protection.

Most relevant to the context of this study is that IL-10 performed better than IL-17 in ROC analysis. The area under the curve AUC of IL-10 and IL-17 was 0.995 with the most optimal diagnostic accuracy compared to other proinflammatory cytokines, and likewise for IL-17 with high sensitivity but low specificity. This difference could signify a limited correlation of IL-10 with the systemic immunological profile of clinically important *E. histolytica* infection in this population. IL-10 has clinical relevance and elevates four-fold in invasive or dysenteric disease, (other markers likely will be more stable than those currently used for the same purpose to

discriminate between patients and controls with the exception of unopsonized *S. dysenteriae* type 1 which itself perdurs). IL-17 had the opposite profile from IL-18, where we would expect an association with inflammation and host defence rather than gut inflammatory conditions suggesting slightly greater biological variability. These findings have practical implications. In circumstances where immunology can supplement classic microscopy that is too non-sensitive to definitively differentiate *E. histolytica* from pathogenically irrelevant morphologically similar species, they can be useful. Importantly, serum IL-10 specifically may be a useful biomarker of disease severity and to help differentiate diagnostic categories, while IL-17 likely provides more information on appropriate inflammatory activation related to progressive disease. However, cytokine measurements should be viewed as adjunctive rather than replacement tools for estimating disease-intrinsic factors, especially since host immune responses may differ based on age, nutritional status, microbiota composition, coinfections and adaptive immune regulation. The current findings should also be considered in light of some limitations. The sample size was relatively small, particularly once stratifying the patients into severity subgroups (which may diminish the precision of subgroup comparisons). The temporal dynamics of IL-10 and IL-17 during the various states of infection, treatment, and recovery could not be assessed because cytokines were measured at one time point. Third, optimal diagnostic performance needs to be externally validated in a larger and independent cohort prior to clinical implementation. Lastly, the study investigated only two cytokines, while it is well known that amebiasis pathogenesis involves a more complex network of inflammatory and regulatory mediators (IFN- γ , TNF- α as well as IL-4, IL-6 and mucosal immune effectors).

Despite these limitations, the present findings contribute meaningful evidence that immune profiling can help clarify the host response to intestinal amebiasis. The stepwise increase of both IL-10 and IL-17 with disease severity supports their biological relevance in the progression of *E. histolytica* infection, while the outstanding ROC performance of IL-10 highlights its potential value as a clinically informative biomarker. Further large-scale and longitudinal studies are needed to validate optimal cutoff values, assess treatment-related changes, and determine whether combining cytokines with conventional parasitological or molecular assays can improve the diagnosis and prognostic evaluation of amebiasis.

CONCLUSION

In the present study, alterations in both immunoregulatory (IL-10) and pro-inflammatory (IL-17) cytokine responses were associated with increasing clinical severity of *E. histolytica* infection. The

simultaneous increase of these cytokines may show differences in how the host's immune system responds during a sudden amoebic infection. In addition, the excellent diagnostic performance observed for both cytokines supports their potential utility as adjunct immunological biomarkers for disease severity assessment in amoebiasis. These findings provide a basis for future research to clarify the biological significance of their concomitant elevation and to evaluate their clinical applicability in larger patient populations. In summary, these results expand knowledge of host immune responses in amoebiasis and provide a rationale for future studies of cytokine-based immunological markers for clinical disease assessment.

Strengths and Limitations

Strengths

There are some notable strengths of this study. This used a case-control design, restricted to laboratory-identified *Entamoeba histolytica* infection patients and healthy controls, allowing comparison of immune factors in people with disease versus non-disorder persons. Secondly, such infections were categorized into 3 groups which included; mild, moderate and severe clinical outcomes thereby making measurements of the cytokine levels be against severity of disease rather than only infection. Another advantage of the current study design is that in terms of modeling immunopathogenesis of intestinal amebiasis, both cytokines (IL-10 and IL-17) which were analyzed play a major role in regulating T cell function [68]. Measuring them by quantitative sandwich ELISA allowed us to develop a standardized approach to the laboratory, and leveraging ROC curve analysis was essential due to its ability to investigate the diagnostic capability of individual markers. Lastly, this finding supports a level of translatability based on the strong AUC seen for IL-10 and also with IL-17. Furthermore, the study harmonized clinical and laboratory dimensions by associating cytokine concentrations with a symptom-based severity classification. Using this algorithm, we enhanced the clinical interpretability of the data; further supporting a possible application for cytokine profiling in disease stratification and diagnostic discrimination.

Limitations

Some limitations of this study should be considered. The sample size was relatively small, especially when grouping patients into mild, moderate and severe groups, which may affect the precision of subgroup comparisons. Furthermore, cytokines were measured at a single time point only. Hence changes in IL-10 and IL-17 over time during infection, treatment or recovery cannot be determined. In conclusion, although IL-10 and IL-17 have shown promising diagnostic performance, the results and proposed cut-off values should be validated in larger independent cohorts before clinical application.

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