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Article

Evaluation of Some Interleukins In Iraqi Patients With Giardia Infection

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Abstract: The purpose of the current investigation is to ascertain how cellular responses contribute to reducing intestine invasion by Giardia. The study team was made up of 40 females and 20 males, aged 16–70, infected with Giardia intestinalis. Laboratory tests (colonoscopy, stool examination) were used to make the diagnosis. Blood was obtained for analysis both before and two weeks following the conclusion of antiparasitic therapy. There were twenty men and twenty-two women in the control group, aged 18 to 55 years. Serum concentrations of IL-5, IL-13, and IFN-ɣ. were measured using Quantikine human assays. Individuals infected with Giardia intestinalis exhibited a statistically significant elevation in IL-13 and IFN-ɣ levels. The levels of these indicators were not lowered by the antiparasitic medication that was supplied.

Keywords: giardia lamblia, IL-13, IL-5, IFN-ɣ, giardiasis

1. Introduction

Both parasitic antigens and exotoxins can produce local or systemic inflammatory responses in response to parasitic infestations. Eosinophils are the effector cells in antibody-dependent cell cytotoxicity (ADCC), which is the main antiparasitic defensive mechanism. A few strategies that block the parasite's surface receptors, activate the complement system to directly harm the parasite, or boost IgE production are all part of antibody-dependent antiparasitic immunity. When Jimenez investigated the impact of G. intestinalis excretory and secretory antigens, they found that IgE levels were up overall and that IgA secretion was stimulated both locally and systemically. IgA was previously demonstrated to be crucial for G. intestinalis invasion by Zhou et al. [1] [2].

According to certain authors, mast cells release kinin proteinase, prostaglandins, and cell mediators in respond to the antigen of Giardia, while T cells don't produce any cytokines related to lymphocytes [3]. It has been demonstrated that significant invasiveness of G. intestinalis occurs in cases of IgG and IgA deficiency, whereas giardiasis can occasionally be asymptomatic (carrier state) [4].

Also, IL-5 induces a humoral response. Th2 lymphocytes generate this cytokine, which aids in B- and T-cell differentiation and growth induction. Eosinophil precursors are stimulated by IL-5 to proliferate, differentiate, degranulate, and produce reactive oxygen compounds. It causes eosinophilia and has a chemotactic impact on eosinophils [5].Th2 lymphocytes also release IL-13, a cytokine that affects monocytes and B

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lymphocytes by increasing the proliferation of activated B-cells. Controlling the antiparasitic response is its primary physiological function. It suppresses the produce of proinflammatory cytokines (IL-1, IL-6, IL-8, and TNF) and antibody-dependent cytotoxicity [6].

Antibodies that attach to the parasite surface antigens eliminate G. intestinalis from the digestive system. By producing mucus in the gut, which is essential for parasite clearance, the released inflammatory mediators cause harm to the parasites, speed up intestinal peristalsis, and aid in their eradication [7]. Determining the role of cellular and humoral responses in thwarting Giardia intestinalis invasion was the goal of the current investigation. IFN-γ measurements were used to gauge the cellular reaction, whereas IL-5 and IL-13 levels were used to gauge the humoral response. The study's two primary objectives were to determine whether a G. intestinalis infection affects the parameters being examined and if the antiparasitic treatment changed the immune response.

2. Materials and Methods

60 individuals (aged 16–70 years) with Giardia intestinalis infections were included in the study group; 40 of them were women and 20 were men (G1). From September to December 2024, the patients were admitted to the Tikrit Hospital's Department of Internal Diseases in Salah Aldin province.

The clinical manifestations (diarrhea, acute abdominal pains, weakness, and sub-febrile temperature), parasitological analysis of the feces, and microscopic analysis of the contents of the duodenum were used to identify giardiasis. Cholagogues (cholamide, cholestil), metronidazole (approximately 10 days), tinidazole (two days), and bioregulators of physiological intestinal flora (lakcid) were administered to the patients. Blood was obtained for analysis before therapy (subgroup G1) and two weeks after antiparasitic medication (subgroup G2: 24 patients, 14 women, and 10 men).The study included 42 healthy individuals (between the ages 20–45) in the control group (C), 20 of whom were men and 22 of whom were women. Every parameter that was looked at in both the control group and the research group in terms of gender and age was statistically analyzed. Every patient consented to participate in the trial in accordance with the Guidelines for Good Clinical Practice. Serum levels of IFN-γ, IL-5, and IL-13, and were measured using the ELISA method with a Quantikine human kit (R&D Systems, USA). The Statistica 8.0 software was used to statistically analyze the results. The features compatible with a normal distribution were calculated using the t-Student test.

3. Results

Our analysis showed that the average amount of IL-5 in G. intestinalis (G1)-infected individuals were significantly lower ($p < 0.05$) than those in healthy patients (Table 1). The average amount of IL-5 rose during antiparasitic treatment (G2), although it wasn't as high as in group C, the control (Table 1). The leukocyte and eosinophil counts for the study and control groups were determined to be within the normal range (no data displayed).

Table (1): Immune response measured by IL-5 levels in G. intestinalis-infected individuals.

Parameters	Study of the G1 subgroup n=60 ×± SD	Study of the G2 subgroup n=24 ×± SD	Study of the C subgroup n=42 ×± SD
IL-5	**3.26 ± 1.13**	**3.60 ± 1.39**	**4.58 ± 1.60**

G1:G2 0.2<P<0.3, G1:C P<0.05*, G2:C P<0.05*

"Values of $p < 0.05$ were considered to be significant"

While, The mean IL-13 level was six times greater in G. intestinalis (G1)-infected individuals than in healthy people (group C) ($p < 0.001$). The result in subgroup G1 remained unchanged following antiparasitic treatment (G2) (Table 2).

Table (2): Immune response measured by IL-13 levels in G. intestinalis-infected individuals.

Parameters	Study of the G1 subgroup n=60 ×± SD	Study of the G2 subgroup n=24 ×± SD	Study of the C subgroup n=42 ×± SD
IL-13	**43.03 ± 27.80**	**41.39 ± 31.10**	**7.01 ± 3.89**

G1:G2 0.5<P <0.6, G1:C P <0.001*, G2:C P <0.001*

The average amount of IFN-ɣ in Giardiasis (G1)-infected patients was over three times higher than that observed in healthy subjects (group C). The statistical significance of the difference between G1 and G2 was not established, the level of IFN-ɣ was lower after the antiparasitic treatment (G2) than it was in subgroup G1 (Table 3).

Table (3): Immune response measured by IFN-ɣ levels in G. intestinalis-infected individuals.

Parameters	Study of the G1 subgroup n=60 ×± SD	Study of the G2 subgroup n=24 ×± SD	Study of the C subgroup n=42 ×± SD
IFN-ɣ	**24.89 ± 13.81**	**23.49 ± 12.38**	**7.02 ± 3.89**

G1:G2 0.8<P <0.9, G1:C P <0.001*, G2:C P <0.001*

4. Discussion

The fight against parasitic infestations involves both humoral and cellular processes. The humoral response involves antibodies, but the cellular response involves direct interaction between parasite antigens and T-cells and produced cytokines. It has been shown in animal models that giardiasis can affect the host organism's immunological response [8]. Class IgG and IgA antibody levels are decreased in persons infected with this parasite. When G. intestinalis invades, the immune system responds by producing antibodies and releasing inflammatory mediators, which damages the parasite. As it aids in the removal of this parasite from the digestive system, IgA is crucial. The nonspecific inflammatory reaction's byproducts, TNF and IL-1, stimulate goblet cells and produce mucus, which makes it easier to remove deceased parasites [9], [10].According to the current investigation, the level of IL-13 rose statistically significantly (six times) over the course of giardiasis in comparison to healthy participants. It is well known that IL-13 increases the synthesis of IgE and antiparasitic immunity while also suppressing ADCC and lowering the release of cytokines that promote inflammation, such as TNF, IL-1, IL-8, and IL-6. Numerous writers have mentioned how parasitic invasions alter the levels of immune indices, such as the noted rise in IL-5 that is indicative of parasitosis [11].

Previous studies conducted by the authors on G. lamblia-infected patients showed a statistically significant rise in IL-5 levels [12]. Both IL-5 and IFN-ɣ levels significantly increased during the acute phase of Schistosoma mansoni infection [13]. Brattig et al. [10] found that IL-5 and IL-13 increased when the extract of the soluble antigen of Onchocerca volvulus was given. Cooper et al. [14] reported that the early stages of Onchocerca volvulus infection resulted in elevated IL-5 and IFN-ɣ production. Peripheral blood mononuclear cells (PBMC) produced statistically significant amounts of IFN-ɣ, IL-5, and IL-13 in individuals with chronic Leishmania infection (but without treatment), according to Ajdary et al. [15]. These results point to the possibility of a mixed type Th1/Th2 reaction. Insufficient IL-5 and IL-13 levels and elevated IFN-g concentrations, however, were indicative of a Th1 response in acute illness patients. Ishikawa et al. [16] found that mice

were infected experimentally with roundworms, Nippostrongylus brasiliensis and T. spiralis, which resulted in the release of cytokines from Th1 and Th2 cells, including IL-5 and IFN-ɣ. The production and release of IgE antibodies is inhibited by IFN-ɣ, that is generated by Th1 cells that are activated. IFN-ɣ promotes the intracellular destruction of parasites by phagocytes by triggering the generation of reacting oxygen and hydrogen peroxide release [17].

According to Touil-Boukoffa et al. [18], in addition to IFN-ɣ, IL-6 is also involved in the defence mechanisms during echinococcosis. Ajami and Rafiei's study [19] revealed increased IFN-ɣ, IL-5, IL-12, and IL-13 levels in patients infected with Hymenolepis nana. We discovered that G. intestinalis-infected patients had higher of IL-13 and IFN-ɣ levels. Th1 lymphocytes aid in the cellular type response and generate IFN-g, which suppresses Th2 cell activity and proliferation. Th2 cells stimulate the humoral response and release IL-10, IL-13, IL-5, IL-6, and IL-4. Our results might indicate that an acute inflammatory state is not associated with a G. intestinalis infection. Both the Th1 and Th2 responses appear to be present in giardiasis, according to the current investigation. Chronic inflammation is a side effect of this parasitic invasion, and the antiparasitic medication appears to raise IL-13 levels while having little effect on other parameters.

5. Conclusion

Significant immunization of the host organism is shown by raised IFN-ɣ and IL-13 levels during giardiasis. When treating giardiasis, antiparasitic medication does not cause the parameters under study to return to normal.

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