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Article

Study of Some Immunological Markers in Women Infected with Trichomonas Vaginalis

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Abstract: Background: Trichomonas vaginalis is a flagellated protozoan parasite responsible for Trichomoniasis, impacting the human urogenital tract and ranking among the primary causes of non-viral sexually transmitted infections. The present study aimed to evaluate the effect of T. vaginalis on some immunological parameters in infected women. Methods: The study included 100 samples, of which 70 were suspected of trichomoniasis and 30 were used as a control group. Urine and vaginal fluid samples were collected from women suffering from vaginal discharge. IgG and IgM level with Interleukin and TGF-β level were investigated by ELISA. Results: The results of the current study showed that out of 70 suspected cases of trichomoniasis, 50 (71.4%) were infected with parasites, while 20 (28.6%) of them gave negative results. High percentage of infection was showed at ages 26-40 years with (46%) percentage. Mean level of IgM and IgG was high in patient with T. vaginalis (7.8± 2.1, 9.3± 1.1 ng/dl) compared with healthy group (5.9± 1.3, 6.33±3.1ng/dl). Mean level of IL-10 and IL-8 was high in patient with T. vaginalis (25.9± 6.3, 134.65± 12.3 pg/ml) compared with healthy group (5.9± 1.3, 99.01±9.4 pg/ml). Mean level of Tumor Growth Factor-β was high in patient with T. vaginalis (101.2± 28.2 pg/ml) compared with healthy group (24.6± 8.5 pg/ml) with ($p<0.001$). Conclusion: From these observations it is concluded that parasitological diagnosis combined with immunological evaluation is very useful in the understanding of the pathogenesis of the trichomoniasis.

Keywords: Trichomonas vaginalis, Tumor Growth Factor-β, urogenital tract, ELISA.

Introduction

Trichomonas vaginalis is a flagellated protozoan parasite responsible for Trichomoniasis, impacting the human urogenital tract and ranking among the primary causes of non-viral sexually transmitted infections (STIs), with an annual prevalence of 180 million as per WHO estimates[1]. *T. vaginalis* inhabits the female urethra and vagina as well as the male urethra, seminal vesicles, and prostate. The normal acidity of the vagina (pH 4.0–4.5) inhibits infection during the 4–28 day incubation period. However,

once the parasite enters the vagina, its metabolic processes cause the pH to change to an alkalinity of 5–6, which encourages the parasite's growth and duplication [2,3].

Female trichomoniasis symptoms might range from nonexistent to a severe acute inflammatory illness. Common symptoms include a foul-smelling, purulent vaginal discharge along with burning, itching, painful urination, and discomfort during sexual activity. In affected females, physical examinations show vulvitis and vaginitis together with a frothy, yellow-green mucopurulent discharge [4]. Nonetheless, infections may result in serious health issues, including cervical erosion, preterm delivery, and infertility in both genders[5]. These infections are not self-resolving and cause non-ulcerative inflammation of the genital epithelium that may progress to necrosis and haemorrhage. Recent research has connected infection with this parasite to increased susceptibility to HIV, cervical cancer, and aggressive prostate cancer [6,7].

The interaction between host and parasite in trichomoniasis is intricate. It depends on the host's immunological response to trichomoniasis and the pathogenic factors of the parasite[8]. After strong cellular attachment to epithelial cells and successful target cell destruction and phagocytosis, the parasite breaches the long-lasting physical barrier of the female reproductive system, causing harmful consequences and a permanent infection. The inflammatory host's T cells' active or dormant immune response is essential for controlling or exacerbating trichomoniasis [9]. Cytokines, together with interleukins, form a diverse collection of proteins, including peptides and glycoproteins, secreted by specific immune system cells. They are produced throughout the entire framework with the aid of diverse immune cells[10]. The present study aimed to evaluate the effect of T. vaginalis on some immunological parameters in infected women.

Materials and Methods

Study design

The study included 100 samples, of which 70 were suspected with trichomoniasis and 30 were used as a control group. Urine and vaginal fluid samples were collected from women suffering from vaginal discharge. The ages of the women attending ranged from 17 to 60 years, during the period from January to May 2026.

Collection of Samples

Urine samples were collected from women with vaginal discharge in sterile test tubes. Vaginal fluid samples were also collected using cotton swabs to obtain samples from the posterior vaginal fold using a speculum. These swabs were placed in their own tube containing 0.5 ml of sterile saline solution immediately after sampling. The sample was stored at 27°C and examined within a few hours of collection[11].

Vaginal pH measurement

Vaginal pH was measured using pH paper by placing it in contact with a sample of vaginal fluid and comparing the resulting color to the standard colors listed on the pH box leaflet, or by placing it in contact with the tip of the speculum after withdrawal. Contact with the cervical mucosa, menstrual blood, or semen should be avoided, as this will give false results. pH is commonly used as an indicator of infection

Urine Examination

Urine samples were collected in sterile test tubes and centrifuged at 1000 rpm for 1 minute for five minutes. The supernatant was then transferred to another container. A drop of the deposit was placed on a clean glass slide, covered with a slide cap, and examined under maximum magnification (40x) to observe purulent cells and epithelial cells, as well as to investigate the presence of parasites[12].

Wet Preparation Slide

A drop of saline solution was placed on a clean, sterile glass slide. The sample was removed from the cotton swab by rolling the swab handle across the slide and mixing it thoroughly with the saline solution. The slide cap was then placed over the slide and examined under maximum magnification (40x) to detect purulent cells, epithelial cells, and parasites.

Collection of Blood Samples

Blood samples were collected for the aforementioned period. 5 ml of venous blood was withdrawn from both the infected and control women using a wine syringe. The blood samples were placed in a

test tube for coagulation at room temperature (20-25°C) for 5 minutes. The serum was then separated using a centrifuge at 3000 rpm. The serum was divided into five portions, each used for a single assay of the specific laboratory immunoassays. Each portion was placed in 0.5 ml Eppendorf tubes and the samples were stored frozen at -20°C until subsequent laboratory tests, which were performed in an external laboratory.

Diagnosis of Trichomonas vaginalis infection

The commercial assays from SUNLONG/China were used to detect specific antibodies to Trichomonas vaginalis, IgG and IgM in human serum using the ELISA method.

Measurement of immunological parameters

The level of IL-10, IL-8 and TGF-β was assessed by using ELISA kits from SUNLONG/China following manufacturer instruction.

Statistical analysis

The data were statistically analyzed using IBM SPSS Version 22 and the t-test was used. The arithmetic means (mean) and standard error means of the patient and healthy samples were compared at a significance level of less than or equal to p.value ≤ 0.05.

Results

Rate of Trichomonas vaginalis infection

The results of the current study showed that out of 70 suspected cases of trichomoniasis, 50 (71.4%) were infected according to the results of the urine and vaginal samples, while 20 (28.6%) of them gave negative results.

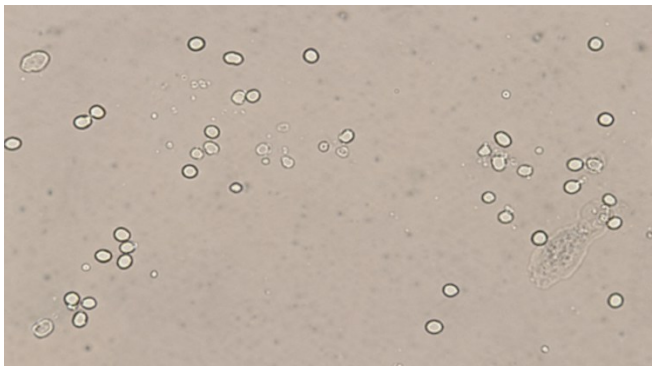


Figure 1. *T. vaginalis* in vaginal discharge smear

Distribution of *T. vaginalis* positive women by age groups

The results in table (1) showed distribution of *T. vaginalis* infection by ages. High percentage of infection was showed at ages 26-40 years with (46%) percentage, followed by 17-25 years with (34%) while the low percentage (20%) was scored in age group (41-60 years).

Table 1. Distribution of *T. vaginalis* positive women by age groups

Ages group	Number	Percentage (%)
17-25	17	34%
26-40	23	46%
41-60	10	20%
Total	50	100%

Mean immunoglobulin levels in *T. vaginalis* infected women and control group

The results in table (2) showed a significant difference in mean level of IgM and IgG between studied groups. Mean level of IgM was high in patient with *T. vaginalis* (7.8± 2.1 ng/dl) compared with healthy group (5.9± 1.3 ng/dl). Also, IgG mean level was high in patient with *T. vaginalis* (9.3± 1.1 ng/dl) compared with healthy group (6.33±3.1ng/dl) with (p=0.001).

Table 2. Mean immunoglobulin levels in *T. vaginalis* infected women and control group

Immunoglobulin	Patients Mean ±SD	Control group Mean ±SD	p-value
IgM (ng/dl)	7.8± 2.1	5.9± 1.3	0.001
IgG (ng/dl)	9.3± 1.1	6.33±3.1	0.001

Mean Interleukins levels in *T. vaginalis* infected women and control group

The results in table (3) showed a significant difference in mean level of interleukins between studied groups. Mean level of IL-10 was high in patient with *T. vaginalis* (25.9± 6.3 pg/ml) compared with healthy group (5.9± 1.3 pg/ml). Also, IL-8 mean level was high in patient with *T. vaginalis* (134.65± 12.3 pg/ml) compared with healthy group (99.01±9.4 pg/ml).

Table 3. Mean Interleukins levels in *T. vaginalis* infected women and control group

Immunoglobulin	Patients Mean ±SD	Control group Mean ±SD	p-value
IL-10 (pg/ml)	25.9± 6.3	5.9± 1.3	p<0.001
IL-8 (pg/ml)	134.65± 12.3	99.01±9.4	0.009

Mean TGF-β levels in *T. vaginalis* infected women and control group

The results in table (4) showed a significant difference in mean level of TGF-β between studied groups. Mean level of TGF-β was high in patient with *T. vaginalis* (101.2± 28.2 pg/ml) compared with healthy group (24.6± 8.5 pg/ml) with (p<0.001).

Table 4. Mean TGF-β levels in *T. vaginalis* infected women and control group

Parameter	Patients Mean ±SD	Control group Mean ±SD	p-value
TGF-β (pg/ml)	101.2± 28.2	24.6± 8.5	p<0.001

Discussion

Numerous local research showed that our community had a similar trichomoniasis incidence. Both the general public and health officials should be made aware of this significant public health issue. Millions of men, women, and children suffer from serious physical and psychological consequences as a result of sexually transmitted diseases, which are a major source of acute illness, infertility, long-term impairment, and mortality worldwide [13]. The growth in incidence throughout the years and in this research may be due to the absence of a proven and viable prophylactic method for trichomoniasis. Because of the potential bad effects and clinical failures associated with the therapeutic medication of choice, metronidazole and the reemergence of resistant strains novel strategies are needed for the control of T. vaginalis infection, including vaccine manufacturing [14].

The study showed high mean of IgM in *T. vaginalis* patients, this result is in line with the findings of Al-Dulaimi et al., [15], who discovered that the serum of infected patients with the parasite had a higher level of IgM than the control group. Al-Mamoori et al. [16] also found that the serum of women infected with the parasite exhibited elevated IgM levels compared to the control group, indicating that acute T. vaginalis infection activates the humoral immune response, as IgM serves as a marker for the acute phase of infection. Moreover, this leads to revelations and patterns for managing or averting parasitic infections.The findings of the present investigation indicate a notable increase in serum IgG levels in women infected with T. vaginalis relative to the control group. This result aligns with the research conducted by Hamad et al., [17] which shown a rise in IgG levels in the serum of women infected with

the parasite relative to the control group. In addition to the previously discussed factors, immunoglobulin, especially IgG, is crucial in humoral immunity for managing parasitic infections.
Cytokine responses in many microbial illnesses have been documented with differing findings, contingent upon the unique immune response patterns elicited by particular bacteria and their specific evasion mechanisms against immunity[18].
In the present study, we found that the level of IL-8 in the serum of women diagnosed with the infection of *Trichomonas vaginalis* was significantly higher than that of healthy women. This rise is an evidence of involvement of inflammatory mechanisms in the host response to Trichomoniasis. The current results are consistent with a recent study from Iraq that showed high levels of IL-8 in women with *T. vaginalis* compared to women without *T. vaginalis* infection in Duhok, Iraq[12]. Likewise, in a recent experimental investigation carried out by et al., [19], human ectocervical epithelial cells were exposed to T. vaginalis, causing the secretion of more IL-8, indicating the capability of this parasite to activate inflammatory responses in the female genital tract.
Also, results is consistent with research conducted by Nam et al.[20]The present investigation indicated elevated IL-8 levels in infected patients compared to their non-infected counterparts. The elevated levels of IL-8 are attributed to the chemotactic factors and biochemical cues that stimulate neutrophils and activated macrophages to generate additional IL-8 during T. vaginalis infection, aiding in the synthesis of antibodies against this parasite. This is comparable to a study conducted by Ali [21] found that white blood cells release IL-8 which acts against this parasite infection.
The present study showed that women with T. vaginalis had more serum IL-10 than the healthy women. This finding suggests the activation of immunoregulatory mechanisms during parasitic infection. The present finding is consistent with that of Farhan et al.,[22] who found that IL-10 serum levels were significantly higher in women with T. vaginalis infection than among healthy women in a case-control study in Diyala, Iraq. Moreover, in their review on host–parasite immune interactions, Galego and Tasca[23] reported that T. vaginalis, and its secreted products, can alter the production of IL-10 and the responses of immune cells, as seen in the present study.
The higher level of IL-10 could be linked to the host's attempt to control over-activation of inflammation by the parasite. IL-10 is a key anti-inflammatory cytokine secreted by a variety of immune-cell subsets, such as regulatory T lymphocytes and macrophages. Inhibits the production of pro-inflammatory mediators, thereby dampening the activity of pro-inflammatory cells. T. vaginalis contains immunomodulatory components which could play a role in regulating these pathways, interestingly. The protein α-actinin-2 of this molecule has been experimentally linked to the induction of tolerogenic dendritic cells and the increase in IL-10 production by regulatory T-cell populations. This immunomodulatory activity could play a role in creating an environment in which excessive inflammation is prevented, but also might be responsible for blocking effective killing of parasites. Thus, the higher levels of IL-10 that were seen in the present study might represent a regulatory mechanism used by the host as well as a regulatory mechanism used by the parasite to promote immune tolerance and persistence [23,24].
The results of the present study showed that the serum TGF-β level was significantly higher in women infected with *T. vaginalis* than in healthy controls. This discovery indicates that trichomoniasis could also be linked to the activation of immunoregulatory pathways involved in the course of infection, besides the inflammatory response. While there are limited clinical studies that directly examine the levels of circulating TGF-β in women with trichomoniasis, experimental evidence indicates that there may be a connection between TGF-β and immune regulation in women with T. vaginalis. JALOOB ALJANABY et al., [25] showed that the parasite's α-actinin-2 protein can induce immunoregulatory responses through regulatory T lymphocytes and led to increased production of TGF-β in the inducible regulatory T-cell populations. The findings here provide experimental evidence for the role of mechanisms involving TGF-β in the host response to T. vaginalis.
Increased TGF-β levels seen in the present study may be due to its vital functions in regulating inflammatory reactions and immune homeostasis. TGF-β is a multifunctional cytokine that regulates T-cell differentiation, specifically the differentiation and activity of regulatory T lymphocytes. It also helps to regulate excessive inflammatory responses, immune-cell activity, and tissue repair after inflammatory damage. Persistent antigen exposure of the genital mucosa due to the parasite activity in

the trichomoniasis system may lead to a promotion of regulatory immune responses, leading to enhanced production of TGF-B[26]. In addition, the expression of both IL-10 and TGF-β was elevated in the current study, which may be the activation of complementarity immunoregulatory pathways that aim to curb inflammation and minimize tissue damage. But high levels of regulation can also dampen effective anti-parasitic immunity, leading to conditions which allow parasites to persist. However, this is a potential mechanism that warrants additional investigation, especially in determining the amounts and types of regulatory T cells in the genital tract and the levels of cytokines.

Conclusion

The results of the present study show that *T. vaginalis* is an important pathogen in the genital tract infection in the women examined and is important to make accurate diagnoses and provide appropriate clinical management. The infection was seen in all reproductive age groups and so susceptibility does not seem to be confined to a particular age group. Also, there were significant differences in humoral immune responses between infected and non-infected women, with higher levels of IgM and IgG in the former, indicating activation of the immune system in reaction to the parasitic infection. Both inflammatory and immunoregulatory systems appear to be activated during the host response to *T. vaginalis* as reflected by the increased levels of IL-8 and IL-10. Moreover, the significant rise in TGF-β provides a possible insight into regulatory immune mechanisms which can modulate the inflammation during infection. Altogether, these results highlight the complexity of the relationship between the T. vaginalis and the immune system of the host in which antibody mediated contact as well as inflammatory cytokines and immunoregulatory factors are involved. From these observations it is concluded that parasitological diagnosis combined with immunological evaluation is very useful in the understanding of the pathogenesis of the trichomoniasis.

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