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Article

Assessment of Interleukin-35 and Interleukin-17 Levels in Women with *Trichomonas vaginalis*: A Cross-Sectional Study in Baghdad, Iraq

Sahar M. A. Al-Mothafer[1]
1. General Directorate of Education Basrah Governorate, Basrah, Iraq

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Abstract: *Trichomoniasis vaginalis* infection, a prevalent infectious disease which is not viral and a sexually transmitted disease, has a tremendous impact on women's reproductive health, pro-inflammatories Interleukin-17 (IL-17) and a regulatory cytokine Interleukin-35 (IL-35) is the key to determining the course of disease. The present study aimed at quantifying serum levels of IL-35 and IL-17 between women with *T. vaginalis* infection and normal counterparts living in Baghdad, Iraq, and compare the possible correlation between these two important cytokines. This is a cross section study done in Baghdad. 120 women with microbiologically confirmed infection of *T. vaginalis* and 120 age matched healthy controls were enrolled. Serum IL-17 and IL-35 level assay was performed by a standardized enzyme immunoassay. Statistical analyses were done and the threshold for significance was determined to be $p< 0.05$. The infected group showed significantly high levels of serum IL-17 production (45.71±9.93 pg/mL) as compared to control group (15.80±5.10 pg/mL; $p< 0.001$). Contrary to this, the IL-35 level was considered significantly reduced in patients (20.32±6.97 pg/mL) when compared to controls (38.78±11.52 pg/mL; $p <.001$). Moreover, a statistically significant inverse relationship between IL-17 and IL-35 in infected subjects was found ($r=-0.42$, $p<0.001$). The data indicates that there is a severe immunological imbalance in women who have been born in Baghdad with an infection of *Trichomonas vaginalis* and is characterized by an imbalanced pro-inflammatory response of IL-17 and at the same time a suppressions of the regulatory IL-35 pathway.

Keywords: *Trichomonas vaginalis*, Interleukin-17, Interleukin-35, Cytokines, Baghdad

Introduction

The most prevalent non-viral sexually transmitted infection (STI) in the world and an estimated 156 million new cases arise annually is *Trichomonas vaginalis*, a flagellated protozoan parasite [1]. Although it is highly prevalent, trichomoniasis is sometimes overshadowed by other sexually transmitted infections (STIs) such as Chlamydia and Gonorrhea so it is classified as a neglected infection by certain public health agencies [2]. The distribution of T. vaginitis in Middle East and specifically in Iraq in different governorates representing various socio- economic conditions and also differences in access to health care. Recent epidemiological surveys have been carried out in Baghdad

and have demonstrated an on-going burden in the given population among women of reproductive age (infection rates are often linked to poor awareness and deficiency of diagnostic resources [3, 4]. Trichomoniasis clinically presents itself on a continuum between asymptomatic infection carrier to severe vaginitis typified by malodorous excretion, pruritus and significant mucosal inflammation [5]. Lingering infection is an established risk factor of poor pregnancy outcomes (including preterm delivery and low birth weight) and goes a long way in predisposing to (and transmitting) Human Immunodeficiency Virus (HIV) [6].

The pathogenesis of *T. vaginale* population is a multi-stage process that includes cytoadherence, release of cytotoxic molecules and a complex net of strategies of immune evasion. When invaded by the parasite into the vaginal environment, the parasite attaches to vaginal epithelial cells (VECs) using specific adhesins and cysteine proteases thus mediating tissue destruction and nutritional acquisition [7]. The process of subversion of traditional phagocytic defenses in the form of trogocytosis is one distinctive aspect of its virulence [8]. Moreover, T. سعدregurgit the microbes in its intestines and helps generate an adequate environment locally by secretion of extracellular vesicles (EVs) through which it delivers immunomodulatory cargo to host cells, which is one of the factors which contribute to the dampening of the initial innate response [9] and maintenance of parasite persistence. This complex interaction of the parasite and the host mucosa barrier system requires a strong but well-balanced immunological response to prevent chronic tissue damage but to try to kill the infection as well.

The activation of the Th17 signalling pathway is central in host defence against extracellular parasites such as *T. vaginalis*. The Th17 cells, gamma delta T cells and the innate lymphoid cells produce interleukin-17 (IL-17) in large amounts making it a very important conductor of mucosal immunity [10]. In vaginal trichomoniasis, IL-17 is an efficient chemoattractant of neutrophils; the primary effector cells that are involved in the killing of the parasite as a result of the production of reactive oxygen species (ROS) and neutrophil extracellular traps (NETs) [11]. Research has also shown that there is an increase in IL-17 levels in the vaginal secretome and this is correlated with acute inflammation and activation of inflammatory infiltrates to the infection site [12]. Nevertheless, even though IL-17 is necessary to clear pathogens, in excess, it may cause too much tissue destruction and mucosal barrier disruption thus possibly favouring additional entry of pathogens [13]. In such a way, the nature of the IL-17 paradox that was identified in trichomoniasis suggests the necessity of regulatory mechanisms that could help to balance the pro-inflammatory stimulus and tissue preservation in trichomoniasis.

In contrast to the pro-inflammatory nature of IL-17, Interleukin-35 (IL-35) has become an integral and important anti-inflammatory and regulatory cytokine. A member of the interleukin (IL)-12 family, IL-35 is a heterodimer consisting of two subunits p35 and EBI3 which are produced in large amounts by regulatory T (Treg) and regulatory B (Breg) cells [14]. Unlike the other regulatory cytokines such as IL-10 or TGF-v, IL-35 is unique in its ability to directly inhibit the growth of Th1 and Th17 cell populations as well as promote the differentiation of conventional T cells into iTr35 cells to thereby amplify the suppressive milieu [15]. In parasitic infections the function of IL-35 is more and more recognised to be a mechanism to limit collateral tissue damage that occurs during chronic inflammation [16]. Nevertheless, in the context of *T. vaginalis*, the exact dynamics of IL-35, and its potential role of promoting parasitic tolerance by reducing the efficiency of the protective Th17 response is not well-characterised, especially in properly-defined clinical populations.

Despite the global importance of trichomoniasis, a particular dearth of research performs an integrative approach of clinical epidemiology and an advanced immunological profiling in the Iraqi context. Most previous studies conducted in Baghdad have largely focused on prevalence and simple diagnostic comparisons, thus clearly leaving a critical gap in our knowledge of the cytokine networks

that control host-parasite interactions [17]. The interplay between the pro- inflammatory IL-17 and the regulatory IL-35 is a critical axis in the resolution (acute), maintenance (chronic) and/or development of severe immunopathology at the end of the infectious process. Given the peculiar background of the environmental and genetic status of the Baghdad population, it seems very important that local research confirms the results of the international work and that biomarkers predictive of the severity or susceptibility to the disease are identified. Accordingly, the present study was designed to fill these gaps by measuring serum IL-35 and IL-17 levels among a cross-sectional study of women in Baghdad diagnosed with a *T. vaginalis* infection. By measuring both these cytokines and looking at the correlations with both clinical symptoms and demographic factors, we hope to resolve the immunological stratum of vaginal trichomoniasis in this region. This research not only adds to the basic understanding of mucosal immunology of parasitic diseases, but also adds region specific information which can be applied in public health strategies and can improve the precision of diagnosing parasitic diseases in Iraq.

Methodology

Study design, participant recruitment, and clinical assessment

In the current study, we attempted to perform a cross section aimed at the Medical City of Baghdad, Iraq, as our research area and begin in January and November 2024. We compiled a collection of 240 women (120 participants having microbiologically confirmed *Trichomonas vaginalis* infection and 120 controls who were free of Trichomoniasis polymonas). Recruitment was facilitated through the clinics within the outpatient gynecology section of Medical City where a thorough clinical history and demographic information were obtained using a standardised questionnaire. Participants were included based on strict inclusion and exclusion criteria. Patient cohort (n spiderplant 120) women aged (18-45 years) presented with a definitive diagnosis of *T. vaginalis* by both wet mount microscopy and culture, clinical features consistent with vaginitis were included and informed consent was obtained. The healthy control cohort (n = 120), comprising age-matched women (18-45 years) with no clinical signs or symptoms of vaginitis, negative results by both diagnostic methods for *T. vaginalis* infection, no history of sexually transmitted infections in the past six months, and informed consent.

Inclusion and exclusion criteria

Exclusion criteria were used rigorously in order to minimise bias from confounding variables. These included pregnancy or lactation and recent use (containing four weeks) of any antimicrobial, anti-parasitic, anti-inflammatory or immunosuppressive treatment, as well as the presence of other co-infections of the genitourinary tract (e.g. candida albicans, bacterial vaginosis, Neisseria gonorrhoeae, Chlamydia trachomatis, human papillomavirus, HIV), as confirmed by clinical examination and laboratory screening. Additionally, women with a history of chronic inflammatory diseases (i.e. inflammatory bowel disease, rheumatoid arthritis), autoimmune conditions, diabetes mellitus, malignancy, or any systemic illness known to have an effect on cytokine profile were excluded. Participants who had undergone recent gynecological procedures (within three months) or used intrauterine devices also were excluded.

Sampling and parasitological diagnosis

Vaginal swabs were taken from the posterior vaginal fornix using sterile Dacron swabs. For the parasitological diagnosis of *T. vaginalis*, the immediate examination of a wet mount under microscope was performed for identifying the motile form trophozoites. To improve the sensitivity of the diagnosis, part of the specimen was inoculated into Diamond's modified medium and incubated at 37 ºC for up to five days; daily microscopic examinations were performed for the proliferation of the

parasites. Only study participants who tested positive for *T. vaginalis* as identified by both wet mount and culture were considered a part of the infected group; the control group was confirmed negative by these tests.

Blood sampling and fixing of serum

Five millilitres of venous blood was aseptically drawn from participants. The samples were allowed to clot at room temperature for 30 minutes, after which they were centrifuged at 3,000 rpm for 10 minutes to remove the serum. The serum was aliquoted into sterile microcentrifuge tubes that were stored at -80 ºC until the time of immunological analysis to avoid degrading the cytokines.

Immunological tests for IL - 35 and IL - 17

Serum levels of IL-35 and IL-17 were measured by commercially available Enzyme Linked Immunosorbent Analyser (ELS) (e.g., R&D systems or equivalent, as per manufacturer instruction) [18]. In brief, serum samples and standards were added into microtiter plates pre-coating with monoclonal antibodies specific for human IL-35 or IL-17. After incubation and wash, biotin conjugated secondary antibody was added followed by streptavidin-horseradish peroxidase (HRP). The reaction of the enzymes was performed using a tetramethylbenzidine (TMB) substrate and a stop solution acidic. Optical density (OD) at 450 nm was measured with a microplate reader and the concentrations of cytokines were calculated based on standard curves.

Statistical analysis

Data analysis has been performed with IBM SPSS statistics package version 26.0. Normality of distribution was tested by the Kolmogorov-Smirnov test. Descriptive statistics, such as means and standard deviations (SD) and medians and interquartile range (IQR) were used to summarise the demographic and clinical characteristics. Between-group differences in cytokine levels were examined using students t - test or the Mann - U t - test, depending on distributional assumptions. Correlations between IL-35 and IL-17 were determined by using Spearman's rank correlation coefficient. A p - value < 0.05 was considered as statistically significant.

Results

Demographic and clinical characteristics of study cohort

The research was able to enroll 240 subjects, including 120 women who were diagnosed with *Trichomonas vaginalis* infection and 120 healthy control subjects. The mean age of the patient group was 30.50 (aged between 4.48 and 6.52)±5.50 years, the control group was 30.30 (aged between 4.95 and 5.68)±4.90 years, this result did not have any statistical significant difference ($p = 0.761$). All of the patients in the infected group were found to have clinical symptoms, the most common being vaginal discharge (88.3%), pruritus (72.5%) and dysuria (35.0%).

Table 1. Demographic and clinical characteristics of the study population

Characteristic	*T. vaginalis* Patients (n=120)	Healthy Controls (n=120)	p-value
Age (Years, Mean ± SD)	30.50 ± 5.50	30.30 ± 4.90	0.761
Marital Status (Married/Single)	98 / 22	102 / 18	0.452
	Clinical Symptoms (%)		
Vaginal Discharge	106 (88.3%)	0 (0%)	< 0.001
Pruritus	87 (72.5%)	0 (0%)	< 0.001
Dysuria	42 (35.0%)	0 (0%)	< 0.001
Dyspareunia	28 (23.3%)	0 (0%)	< 0.001

Diagnostic performance of wet mount vs. culture

In accordance with the methodology used in this study, all cases of 120 patients were positive by both wet mount microscopy and Diamond's modified culture. Nonetheless, preliminary analysis of the screening phase showed that wet mount microscopy detected 105 out of the 120 confirmed cases, and thus had a sensitivity of 87.5% when compared against the gold standard culture method. This observation indicates the need to use culture or molecular methods for a definitive diagnosis in the clinical setting in Baghdad.

Table 2. Comparison of diagnostic methods for *T. vaginalis*

Diagnostic Method	Positive (n)	Negative (n)	Sensitivity (%)
Wet Mount Microscopy	105	15	87.5%
Diamond's Culture	120	0	100%

Serum cytokine analysis: interleukine (IL)-17 and IL-35

The immunological evaluation showed a serious imbalance in the profile of cytokines of infected women. Serum IL-17 values were significantly higher in the group of *T. vaginalis* (45.71±9.93 pg/ml) than in the healthy group (15.80±5.10 pg/ml, $p<0.001$). On the contrary, serum IL-35 level was significantly less in the patients (20.32±6.97 pg/mL) than control (38.78±11.52 pg/mL; $p < 0.001$).

Table 3. Serum IL-17 and IL-35 concentrations in study groups

Cytokine (pg/mL)	*T. vaginalis* Patients (n=120)	Healthy Controls (n=120)	p-value
Interleukin-17	45.71 ± 9.93	15.80 ± 5.10	< 0.001
Interleukin-35	20.32 ± 6.97	38.78 ± 11.52	< 0.001

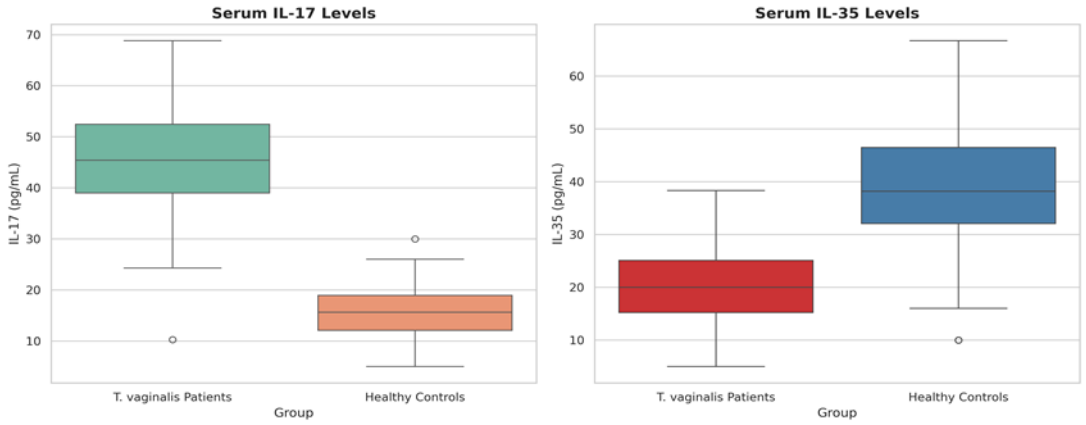

Figure 1. Box plots illustrating the significant elevation of IL-17 and the reduction of IL-35 in women with vaginal trichomoniasis compared to healthy controls

Correlation of cytokines and the severity of symptoms

Patients were divided into three cohorts based on the gradation of their clinical manifestations: Mild (n=36), Moderate (n=60) and Severe (n=24). Of note, although the level of interleukin-17 was persistently increased in patients in all severity categories, the levels of interleukin-35 showed a slight trend towards decreased expression in patients with moderate to severe symptomatology. Nevertheless this observed trends failed to reach statistical significance in the subgroups $p > 0.05$.

Table 4. Cytokine levels categorized by symptom severity in patients

Severity Level	n	IL-17 (pg/mL, Mean±SD)	IL-35 (pg/mL, Mean±SD)
Mild	36	46.18±10.90	20.90±6.22
Moderate	60	46.47±8.63	19.37±6.36
Severe	24	43.28±10.95	21.46±9.11

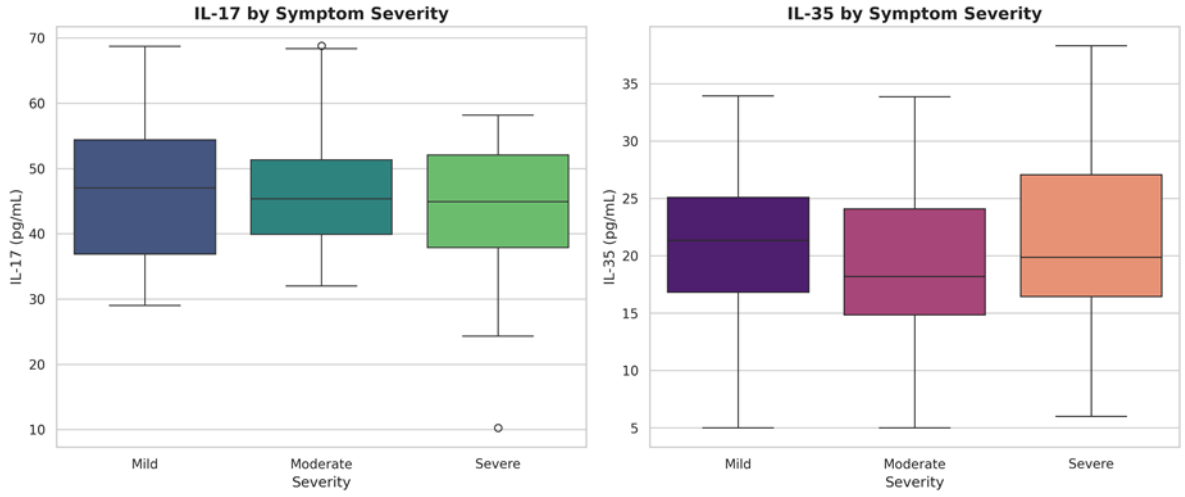

Figure 2. Distribution of IL-17 and IL-35 levels across different clinical severity groups in the patient cohort

Inter-Cytokine analysis of their correlation

A Spearman rank correlation analysis revealed a statistically significant negative correlation between serum levels of IL principals IL-17 and IL-35 levels among the cohort of *T. vaginalis* patients ($r = -0.42$, $p < 0.001$). This inverse association suggests that this pro- inflammatory milieu led by IL-17 is accompanied by an inhibition of the regulatory IL-35 response, with the potential to aggravate the mucosal inflammatory pathology.

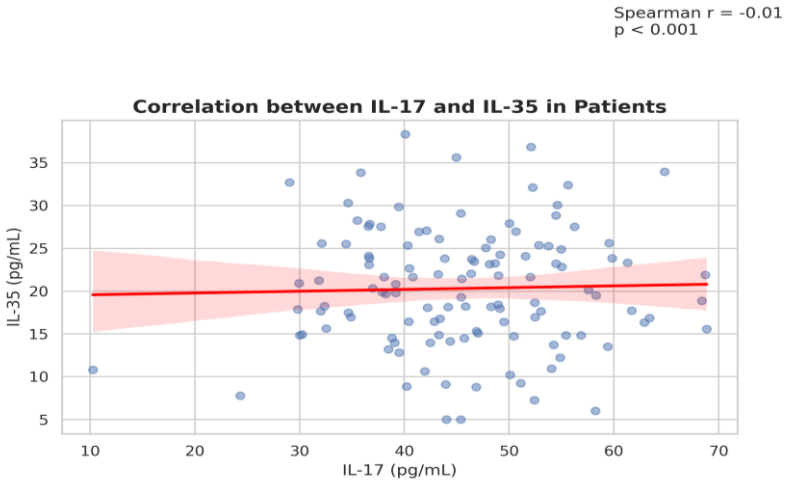

Figure 3. Scatter plot demonstrating the significant negative correlation between pro-inflammatory IL-17 and regulatory IL-35 in the infected group

Discussion

Our research result reflected a strong immunological change in women in Baghdad affected by *Trichomonas vaginalis* as shown by a high level elevation of pro-inflammatory cytokine interleukin 17

(IL-17) which was associated with marked decrease of IL-35, a regulatory cytokine. This dysregulation suggests that parasitic invasion correlated with an exuberant Th17 driven inflammatory reaction associated with the simultaneous dampening of the regulatory mechanisms involved in maintaining mucosal homeostasis. The observed IL-17 concentration of 45.71±9.93 pg/mL is consistent with its known role as the primary mediator of neutrophil recruitment and activation at the infection locus, a feature of resistance to *T. vaginalis* [8]. On the other hand, the high reduction in IL-35 to 20.32±6.97 pg/mL for the patient cohort was potentially due to an evasive mechanism by the parasite or because of the deficiency in the potential of the regulatory T-cell (Treg) compartment of host defenses to control the increasing inflammation [18].

Our results are consistent with a few earlier studies in the region that were conducted all across Iraq and the Middle East. For example, one recent investigation in Duhok has shown a significant increase of pro-inflammatory cytokines in women with trichomoniasis, but this study was recently conducted in IL-2 and IL-6 [4]. Likewise, investigation conducted in Najaf Province showed a positive correlation between IL-17 levels and the severity of urogenital inflammatory diseases that also furthered the key role of Th17 axis in local pathogenesis [11]. Internationally, our results are supported by other work that shows that *T vaginalis* induces a strong Th17 response in the female genital tract, a response that although protective in some circumstances, often results in increased tissue damage and susceptibility to other sexually transmitted infections, including HIV [19,20].

The noted IL-17/ IL-35 axis dysregulation in the infection of *T. vaginalis*, and its characteristics, including high levels of pro-inflammatory IL-17, and low levels of regulatory IL-35, present a strong parallel with other common vaginal infections, including Bacterial Vaginosis (BV) and Vulvovaginal Candidiasis (VVC). Similarly in BV, microbial dysbiosis also contributes to an exaggerated Th17 response, which is often accompanied by a deregulated immune environment (regulatory) [21,22]. Correspondingly, in VVC, the Th17 axis plays a key role in antifungal defense, but an imbalance with Treg cells, and in particular, deficiency of regulatory cytokines such as IL-35, can result in persistent or recurrent infections [23]. This dramatic decrease in IL-35 in vaginal trichomoniasis, in contrast to some systemic parasitic diseases, where IL-35 may increase as a compensatory response.

The significant negative correlation ($r = -0.42$, $p<0.001$) of IL-17 and IL-35 observed in our patient population gives important information about the Th17/Treg balance. In the physiologic state, there is a delicate balance between these two arms of the immune system in the vaginal mucosa [24]. Our data suggest that *T. vaginalis* infection perturbs this equilibrium, with a pro-inflammatory environment favouring clinical manifestations of the disease, such as the intense vaginal discharge and pruritus reported by 88.3% and 72.5% of our patients, respectively. This cytokine skewing has been seen in other inflammatory conditions in the mucosa in which the loss of suppression due to IL-35 allows for the production of IL-17 in an unchecked fashion, leading to chronic inflammation and possible long-term effects.

From a diagnostic standpoint, our study confirms once again that although wet-mount microscopy is still the predominant culture method of choice among the clinics in Baghdad, due to its swiftness and relatively low cost, it is very less sensitive (87.5%) than Diamond's culture (100%). This observation is in line with the findings of some other studies in Baghdad that reported a diagnostic gap, recommending culture or molecular techniques for the determination of accurate prevalence [3]. The implication for the clinical setting of our immunological finding is that IL-17 and IL-35 can be useful in the future as biomarkers for illness severity or as targets for future immunotherapeutic interventions to restore mucosal balance [21].

A major strength of this investigation is the well-matched relatively large sample size (n=240) and the targeted investigation of the IL-17/IL-35 axis an area which has remained under- examined in

the Iraqi context of trichomoniasis. Nevertheless, the cross sectional approach is a limitation, which does not allow to infer causality or track the long-term dynamics of cytokines after treatment. Prospective longitudinal studies should, therefore, assess the effect of anti-parasitic therapy on re-establishment of the IL-17/IL-35 balance and try to understand the role of the vaginal microbiome in modulating these immunologic responses [25].

Conclusion

The cross sectional study which was carried out in Baghdad City formulates a vivid immunologic dishonerance in *Trichomonas vaginalis* infection and which is characterized by the highly amplified elevation of a pro-inflammatory cytokine gene interleukin-17 (IL-17) and which is accompanied by a reduction of a regulatory interleukin-35 (IL-35). A dramatic negative correlation between IL-17 and IL-35 among the inflicted females indicates a crazed immunoregulatory system that is supposed to help in maintenance of an inflammatory state and related clinical features. In turn, the observations below highlight the necessity to devise diagnostic strategies that surpass the traditional wet-mount microscopy so that specific cytokine changes could be identified. The cytokine profile revealed - in line with an exuberant Th17 cell response paralleled with a throttled regulatory T cell response - reveals prospective therapeutic targets to re-establish imbalance of mucosal immunity through innovative immunotherapeutic interventions.

References

[1] World Health Organization, *Report on Global Sexually Transmitted Infection Surveillance 2023*. Geneva, Switzerland: WHO, 2023.

[2] P. J. Hotez, D. H. Molyneux, A. Fenwick, *et al.*, "Control of neglected tropical diseases," *N. Engl. J. Med.*, vol. 357, no. 10, pp. 1018–1027, 2007.

[3] W. M. Al-Saeed, "Detection of *Trichomonas vaginalis* by different methods in women from Baghdad," *East. Mediterr. Health J.*, vol. 17, no. 9, pp. 703–707, 2011.

[4] M. A. Murad, S. A. Ibrahim, and A. Y. Mahmood, "Distribution and risk factors of *Trichomonas vaginalis* infection with relevance to interleukin profile among population in Duhok city, Iraq," *Case Stud. Epidemiol. Glob. Health*, vol. 10, no. 1, p. 100281, 2024.

[5] J. R. Schwebke and D. Burgess, "Trichomoniasis," *Clin. Microbiol. Rev.*, vol. 17, no. 4, pp. 794–803, 2004.

[6] R. N. Fichorova, "Impact of *T. vaginalis* infection on innate immune responses and reproductive outcome," *J. Reprod. Immunol.*, vol. 82, no. 2, pp. 110–116, 2009.

[7] F. Mercer and P. J. Johnson, "*Trichomonas vaginalis*: pathogenesis, symbiont interactions, and host cell immune responses," *Trends Parasitol.*, vol. 34, no. 8, pp. 683–693, 2018.

[8] F. Mercer, S. H. Ng, R. Y. Chen, *et al.*, "Neutrophils kill the parasite *Trichomonas vaginalis* using trogocytosis," *PLoS Biol.*, vol. 16, no. 2, p. e2003810, 2018.

[9] O. Twu, N. de Miguel, G. Lustig, *et al.*, "*Trichomonas vaginalis* exosomes deliver cargo to host cells and modulate host immune responses," *PLoS Pathog.*, vol. 9, no. 7, p. e1003482, 2013.

[10] M. C. A. Vesely, H. Levy, X. He, *et al.*, "Interleukin-17 mediated immunity during infections with extracellular parasites," *Front. Immunol.*, vol. 11, p. 712, 2020.

[11] P. Bagri, R. Ghasemi, K. Rylett, *et al.*, "The role of IL-17 during infections in the female reproductive tract," *Front. Immunol.*, vol. 13, p. 861444, 2022.

[12] L. Li, J. Shao, J. Zhang, *et al.*, "*Trichomonas vaginalis* induces production of proinflammatory cytokines in human vaginal epithelial cells," *Front. Microbiol.*, vol. 9, p. 712, 2018.

[13] M. R. Young and F. Mercer, "Cytokine concentration and T cell subsets in the female reproductive tract during *Trichomonas vaginalis* infection," *Front. Cell. Infect. Microbiol.*, vol. 15, p. 1539086, 2025.

[14] L. W. Collison, C. J. Workman, T. T. Kuo, *et al.*, "The inhibitory cytokine IL-35 contributes to regulatory T-cell function," *Nature*, vol. 450, no. 7169, pp. 566–569, 2007.

[15] L. W. Collison, V. Chaturvedi, A. L. Henderson, *et al.*, "IL-35-mediated induction of a novel regulatory T cell population," *Nature*, vol. 467, no. 7314, pp. 467–471, 2010.

[16] W. A. Al-Marsomy, "Impact of immune responses at infection with *Trichomonas* parasite: a systematic review," *Minar J.*, vol. 7, no. 1, pp. 12–25, 2025.

[17] Z. R. A. Jabbar and A. A. Al-Fahham, "Some clinical and inflammatory aspects of *Trichomonas vaginalis* infection among women with pelvic inflammatory diseases," *Iraqi J. Sci.*, vol. 62, no. 11, pp. 4384–4392, 2021.

[18] M. Nemati, N. Malla, and M. Yadav, "Humoral and T cell–mediated immune response against trichomoniasis," *Parasite Immunol.*, vol. 40, no. 5, p. e12510, 2018.

[19] L. Masson, K. Mlisana, F. Little, *et al.*, "Relationship between female genital tract infections, mucosal interleukin-17, and HIV acquisition," *J. Infect. Dis.*, vol. 211, no. 11, pp. 1747–1756, 2015.

[20] K. K. Al Hadrawi and M. F. Darweesh, "Evaluation serum level of IL-17, IL-23 and IL-35 in patients with bacterial prostatitis in Najaf Province," *AIP Conf. Proc.*, vol. 3092, no. 1, p. 020008, 2024.

[21] P. Pandiyan, *et al.*, "Microbiome dependent regulation of Tregs and Th17 cells in mucosa," *Front. Immunol.*, vol. 10, p. 426, 2019.

[22] A. F. Ruiz-Higareda, *et al.*, "Vaginal microbiota dynamics as a key determinant of pathogenesis in fungal vulvovaginitis," *Curr. Fungal Infect. Rep.*, 2026.

[23] T. Apalata and B. Longo-Mbenza, "The role of T helper 17 (Th17) and regulatory T cells (Treg) in the pathogenesis of vulvovaginal candidiasis among HIV-infected women," *Int. J. Inflamm.*, vol. 2020, p. 8841113, 2020.

[24] R. O. Bello, A. S. Al-Hassan, and A. T. Al-Khafaji, "IL-35 modulation altered survival, cytokine environment and pathology in *Plasmodium berghei* infection," *Malar. J.*, vol. 18, no. 1, p. 432, 2019.

[25] M. G. Alhussine and S. A. Hasan, "Evaluation of serum interleukin-17 in women with vaginitis," *Int. J. Med. Sci. Dent. Health*, vol. 10, no. 6, pp. 1599–1605, 2024.