Jagua Publication

Journal of Applied Health Sciences and Medicine, 6(2):32–36, 2026. DOI: 10.58614/jahsm625

Journal of Applied Health Sciences and Medicine

Journal homepage: https://www.jpub.org/journal-details.php?journal-id=39

ISSN (E): 2583-1887
Journal of Applied Health Sciences and Medicine

Research Article

Association Between *Trichomonas vaginalis* Infection and Semen Quality in Infertile Men: A Retrospective Analysis

Abbas Ghafil Abbas [1] **and Alaa Hachem** [1*]

[1] *Department of Anatomy, College of Medicine, University of Al-Qadisiyah, Diwaniyah City, Iraq.*
[*] *Corresponding author: alaa.hachem@qu.edu.iq*

Article Info

Keywords: *Trichomonas vaginalis, Male infertility, Semen parameters, Oxidative stress, Reactive oxygen species, Malondialdehyde, DNA fragmentation, Leukocytospermia.*

Received: *13.01.2026;*
Accepted: *10.02.2026;*
Published: *20.02.2026*

Abstract

Background: *Trichomonas vaginalis* is a common sexually transmitted infection, which was found to impact male infertility through inflammation and oxidative stress-mediated sperm impairments.
Objective: To investigate the effect of *T. vaginalis* infection on semen quality, inflammatory semen findings, oxidative stress markers, and sperm DNA fragmentation.
Methods: A retrospective case-control study included 150 infertile men and 150 fertile controls. *T. vaginalis* infection was diagnosed by PCR, and semen parameters were compared between infected and uninfected men. Inflammatory semen indicators of leukocytospermia, viscosity, liquefaction status, and semen culture positivity were extracted from laboratory records. Oxidative stress markers were investigated through measuring reactive oxygen species (ROS) with fluorometric ROS assay (DCFH-DA), malondialdehyde (MDA) with thiobarbituric acid reaction, and sperm DNA fragmentation with TUNEL assay.
Results: Prevalence of *T. vaginalis* among in infertile men was found higher than fertile controls (18.0% vs 4.0%, $p < 0.001$). Infected men also showed lower semen volume (2.2±0.9 vs 2.5±0.7 mL, p=0.040), lower concentration (30.5±13.4 vs 54.3±20.7 million/mL, $p < 0.001$), lower total sperm count (72.4±33.1 vs 135.6±57.2 million, $p < 0.001$), reduced progressive motility (26.8±9.5% vs 46.2±12.3%, $p<0.001$), total motility (38.6±11.4% vs 58.9±14.6%, $p<0.001$), morphology (2.9±1.3% vs 5.1±2.0%, p=0.015), and vitality (55.4±10.2% vs 69.8±11.5%, p=0.024). Cases with leukocytospermia, higher viscosity, delayed liquefaction, and positive semen culture were more frequent in infected men (all $p<0.05$). ROS, MDA, and DNA fragmentations were markedly higher in the infected group (all $p\leq 0.01$).
Conclusion: *T. vaginalis* infection was strongly associated with altered semen characteristics accompanied by higher inflammatory and oxidative stress-related sperm damage.

1. Introduction

It is estimated that almost half of all infertility cases can be related to male factor infertility [1]. However, many of these causes remain mostly unidentified. Research shows that male genital tract infections can play a significant part in male fertility impairment [2]. The infection with *Trichomonas vaginalis* is considered as one of the most common sexually transmitted protozoan infection [3]. In men, the infection is either develops as asymptomatic condition or presents with mild signs, resulting in high proportions of subclinical cases or to find other cases with chronic persistence of the disease [4]. This results in a condition where men could suffer from an undiagnosed genital tract inflammation that have grievous consequences on reproductive ability [5].

How To Cite: A.G. Abbas, A. Hachem. Association Between *Trichomonas vaginalis* Infection and Semen Quality in Infertile Men: A Retrospective Analysis. *Journal of Applied Health Sciences and Medicine*, 6(2):32–36, 2026. https://doi.org/10.58614/jahsm625

Studies showed that *T. vaginalis* infection could negatively impact semen quality through what is proposed as an inflammation-driven oxidative stress [6]. Production of excessive reactive oxygen species (ROS) was found to alter the native cellular mechanisms that govern the seminal antioxidant defenses, resulting in reduced sperm motility, disrupted membrane integrity, and degraded sperm DNA [7]. Several studies have suggested a valid correlation between *T. vaginalis* infection and abnormal semen parameters, presenting lower motility and concentration [6, 8]. In contrast, other studies questioned the impact of *T. vaginalis* on male fertility, describing no significant impact on routine semen findings between infected and non-infected men [5]. Such reported discrepancies between studies might indicate variations in study design, diagnostic approach, sample size, and duration of infection.

Given the prevalence and the questioned impact of *T. vaginalis* on sperm characteristics in addition to the reversable nature of the infection following the appropriate treatment, examining the protozoal infection in a cohort of infertile men is of clinical significance. Therefore, this study aimed to assess the role of *T. vaginalis* infection in male infertility by examining the pathogen prevalence among a cohort of infertile men and the impact of the infection on semen quality, semen oxidative stress, and the contribution to the raised risk of infertility.

2. Materials and methods

The current retrospective case-control study was conducted at the Department of Andrology and fertility at the Teaching Hospital of Diwaniyah City, Iraq. Patients' medical records and laboratory findings were collected and reviewed for those men who were eligible to the study's inclusion criteria for the study period between July 2024 and September 2025. The study was designed to compare between infertile men (cases) with fertile men (controls) for investigating the association between *T. vaginalis* infection and semen quality, inflammatory semen findings, and semen oxidative stress.

Cases of the study were defined as infertile adult men who failed to achieve successful pregnancy after at least 12 months of regular unprotected intercourse. Controls of the study constituted of proven fertile men with confirmed partner pregnancy or fatherhood within recent years, in addition to no history of infertility. The study collected data from 300 participants that were encompassed of 150 infertile men and 150 fertile controls.

Eligible data and inclusion to the study was based on the availability of a complete semen analysis and test result of *T. vaginalis* which should have been obtained at the same time of the semen examination. Patients or controls with missing data were excluded, also all cases with unconfirmed *T. vaginalis*, or in cases when infertility was due to non-infectious causes (e.g., genetic syndromes, dysregulated hormonal profiles, or varicocele). Individuals with chronic systemic diseases, chemotherapy, radiotherapy, exogenous androgen usage were also excluded.

Samples collection of semen was obtained by masturbation inside sterile containers after three days of abstinence, and semen volume in addition to liquefaction time were measured after incubation of semen sample at 37° C for 30 minutes. Semen analysis was performed in accordance with World Health Organization (WHO) laboratory guidelines [9], through which sperm concentration, sperm motility (as progressive and total motility), and sperm morphology were reported. Based on these findings, individuals' semen profiles were categorized into normozoospermia (for semen values matching the reference values set by the WHO) and abnormal semen values that including oligozoospermia, asthenozoospermia, teratozoospermia, and oligoasthenoteratozoospermia, while leukocytospermia was reported positive in cases were more than 1×10^6 of white blood cells found in a milliliter semen sample. *T. vaginalis* infection was confirmed using the PCR diagnostic method employed at the laboratory center of the hospital.

Oxidative stress and sperm DNA integrity outcomes were extracted from laboratory reports. ROS measurements and malondialdehyde (MDA) levels were recorded as performed in routine testing, and DNA fragmentation results were recorded as a percentage.

All statistical analyses were performed on SPSS software (IBM, USA). For comparison between groups independent-samples t test was used to compare between scale variables, whereas chi-square test or Fisher's exact test were used to compare between categorical variables. All statistical significance was defined as $p < 0.05$.

The study protocol was approved by the ethical committee at the College of Medicine- University of Al-Qadisiyah. Due to the retrospective nature of the study, informed patients' consents were waived, while patients' confidentiality was preserved throughout the stages of extraction of data, evaluation, and reporting.

3. Results

Results showed that *T. vaginalis* infection was detected in 27 infertile men (18.0%) compared to only 6 of the fertile controls (4.0%). The finding demonstrates a notably higher prevalence among infertile men ($p < 0.001$) Table 1.

Table 1: Rate of *Trichomonas vaginalis* infection among infertile and fertile individuals

Study group	Total (n)	*T. vaginalis* positive n (%)	*T. vaginalis* negative n (%)	P value
Infertile men	150	27 (18.0%)	123 (82.0%)	<0.001
Fertile controls	150	6 (4.0%)	144 (96.0%)	

The results also revealed a considerable impairment in semen quality in *T. vaginalis*–positive men compared with uninfected individuals. Infected men showed lower semen volume (2.2 ± 0.9 vs 2.5 ± 0.7 mL, $p = 0.040$), reduced sperm concentration (30.5 ± 13.4 vs 54.3 ± 20.7 million/mL, $p < 0.001$), and decreased total sperm count (72.4 ± 33.1 vs 135.6 ± 57.2 million, $p < 0.001$). Progressive motility and total motility were significantly reduced in the infected group (26.8 ± 9.5% vs 46.2 ± 12.3% and 38.6 ± 11.4% vs 58.9 ± 14.6%, respectively; both $p < 0.001$). Normal sperm morphology as well as vitality were also significantly lower among *T. vaginalis*–positive men ($p = 0.015$ and $p = 0.024$, respectively) Table 2.

Table 2: Semen parameters according to *T. vaginalis* infection status

Semen parameter	*T. vaginalis* positive Mean ± SD	*T. vaginalis* negative Mean ± SD	P value
Semen volume (mL)	2.2± 0.9	2.5± 0.7	0.040
Sperm concentration(million/mL)	30.5± 13.4	54.3 ± 20.7	<0.001
Total sperm count (millio	72.4± 33.1	135.6± 57.2	<0.001
Progressive motility (%)	26.8± 9.5	46.2± 12.3	<0.001
Total motility (%)	38.6± 11.4	58.9± 14.6	<0.001
Normal morphology (%)	2.9± 1.3	5.1± 2.0	0.015
Vitality (%)	55.4± 10.2	69.8± 11.5	0.024

Normozoospermia was significantly less common among *T. vaginalis*–positive men compared with uninfected individuals (14.% vs 50.5%, $p < 0.001$). In contrast, asthenozoospermia and oligoasthenoteratozoospermia were more frequent in the infected group. Oligozoospermia and teratozoospermia were also more commonly observed among infected men, although these differences did not reach statistical significance Table 3.

Table 3: Semen abnormalities according to *T. vaginalis* infection

Semen pattern	*T. vaginalis* positive n (%)	*T. vaginalis* negative n (%)	P value
Normozoospermia	4 (14.8%)	138 (50.5%)	
Oligozoospermia	6 (22.2%)	42 (15.4%)	
Asthenozoospermia	8 (29.6%)	36 (13.2%)	<0.001
Teratozoospermia	3 (11.1%)	24 (8.8%)	
Oligoasthenoteratozoospermia	6 (22.2%)	33	

Markers of genital tract inflammation were significantly more frequent in *T. vaginalis*–positive men. Leukocytospermia was detected in more than half of infected individuals (55.6%) compared with 19.8% of uninfected men, ($p < 0.001$). Increased semen viscosity, delayed liquefaction, and positive semen culture results were all significantly associated with *T. vaginalis* infection (all $p < 0.05$) Table 4.

Table 4: Inflammatory semen findings associated with *T. vaginalis*

Parameter	*T. vaginalis* positive n (%)	*T. vaginalis* negative n (%)	P value
Leukocytospermia (1×10^6 WBC/mL)	15 (55.6%)	54 (19.8%)	<0.001
Increased semen viscosity	11 (40.7%)	48 (17.6%)	0.007
Delayed liquefaction	9 (33.3%)	41 (15.0%)	0.019
Positive semen culture	10 (37.0%)	39 (14.3%)	0.003

Men infected with *T. vaginalis* showed considerably higher oxidative stress levels. This was estimated by the elevated ROS and MDA concentrations compared with uninfected men ($p < 0.001$ and p=0.014, respectively). Additionally, sperm DNA fragmentation was found higher in the infected group (34.6 ± 8.9% vs 19.7 ± 6.4 $p < 0.001$) Table 5.

Table 5: Oxidative stress and sperm damage markers

Marker	*T. vaginalis* positive (Mean ± SD)	*T. vaginalis* negative (Mean ± SD)	P value
ROS	3.8 ± 1.1	1.9 ± 0.8	<0.001
Malondialdehyde (MDA)	4.2± 1.3	2.5 ± 0.9	0.014
DNA fragmentation (%)	34.6± 8.9	19.7 ± 6.4	<0.001

4. Discussion

This study found that *T. vaginalis* was significantly more frequent in infertile men than fertile controls (18.0% vs 4.0%, p<0.001), and the protozoan infection was associated with reduced semen quality and higher oxidative damage in the semen. Several studies align with these findings confirming the impact of the *T. vaginalis* infections on male reproduction. A study reported 31.8% of infertile patients among the Iraqi cohort diagnosed with *T. vaginalis*, which was associated with dramatic declines in sperm count and motility [10]. A systematic review on the impact of *T. vaginalis* on male fertility parameters concluded that *T. vaginalis* infection rates are markedly higher in infertile than control groups and highlighted sperm impairment and infection-triggered immune reactions and other inflammatory responses as principal pathways linking infection to infertility [11]. By contrast, Hosseini et al. detected *T. vaginalis* in only 1 of 197 infertile men, providing evident on the impact of geographical and methodological variabilities between studies [5]. It appears that the current prevalence rate allocated between these limits, signifying that the actual infection rates are likely depending on the local STI distribution and diagnostic methods used. Indeed, it has been emphasized that inconsistencies across studies often stem from differences in detection techniques and timing of sampling [12].

The current results showed that infected men with *T. vaginalis* resulted in altered semen parameters than uninfected men. Sperm concentration, progressive motility, and normal morphology were reduced in our cases, while seminal leukocytes and abnormal viscosity were amplified. These observations align with other studies. A study reported that asymptomatic men with *T. vaginalis* had evidently altered

semen results (e.g. lower motility and abnormal morphology) compared to healthy controls [13]. Similarly, Jabr et al. mentioned 46% lower sperm concentration and 68% lower motility in *T. vaginalis*-positive patients [10].

In our study the infected men often displayed leukocytospermia and hyperviscosity, signifying an inflammatory process. Consistent with this, research reported *T. vaginalis* in prostatic and epididymal fluids of infertile men, accompanied by signs of prostatitis and epididymitis [14]. This inflammation can itself impair spermatogenesis or sperm function [15]. Besides, anti-trichomonal therapies often reversed these effects. In asymptomatic patients with *T. vaginalis*, a single metronidazole treatment course was found to normalize semen parameters in roughly half of infertility cases [13], suggesting that the infection was the causative agent. Our semen findings are mostly in agreement with these reports, indicating that *T. vaginalis* infection tends to correlate with poorer semen quality and semen inflammation.

The current infected patients exhibited elevated markers of oxidative stress (ROS, MDA) and higher sperm DNA fragmentation than controls. Similar studies on STI suggests that infection-induced inflammation drives ROS-mediated sperm injury [16]. In cases of chronic viral infections like hepatitis B, the infection was shown to be associated with raised seminal malondialdehyde (MDA) and proinflammatory cytokines in infected men [12]. In vitro analyses confirm that *T. vaginalis* can directly disturb sperm function causing higher sperm DNA fragmentation index (DFI) and altered seminal chemistry [17]. Recent reviews of leukocytospermia and bacteriospermia in male infertility similarly emphasize that inflammatory cells and infection-related oxidative stress can adversely affect functional sperm outcomes [18]. Consistent with this pathway, *T. vaginalis*–positive men presented in our study had almost doubled ROS levels and higher MDA, in addition to higher rate of DNA fragmentation (34.6% vs 19.7%, $p<0.001$). This is likely driven by leukocyte activation in the infected male tract, which would certainly reduce sperm membranes integrity. These findings are in line with the notion that genital tract infections induce oxidative stress, a well-known mechanism of infection-related infertility [16].

Recent clinical studies on the impact of STI on male infertility support a positive association between seminal redox imbalance and sperm DNA fragmentation. For instance, a systematic review found a mutual correlation between seminal oxidation-reduction potential and DNA fragmentation across thousands of semen samples, supporting the role of oxidative stress as a central regulator of DNA damage [19]. The present findings align with the proposed mechanism where *T. vaginalis* and the associated inflammation contribute to oxidative injury and DNA fragmentation, which negatively impact motility, morphology, and vitality.

Although, the exact mechanism through which *T. vaginalis* impairs semen quality is still uncovered, *T. vaginalis* likely impairs semen quality through direct sperm injury. In vitro investigation showed that *T. vaginalis* produce secretory proteins (TvESPs) which were found to significantly reduce sperm survival and motility, damages the acrosome, and trigger sperm apoptosis [6]. Additional proposed mechanism is through infection-associated leukocytospermia, which generate ROS and proinflammatory cytokines. The resulting oxidative burst following the inflammatory process can peroxidize sperm lipids and fragment DNA [20]. Moreover, infection with *T. vaginalis* could results in obstruction and scarring of the male genital tract and the accessory glands associated with the tract, and this is evident in cases of orchitis or epididymitis which results in atrophic testes, reduced testosterone, and azoospermia [13, 21]. Furthermore, patients with *T. vaginalis* may be more likely to be con-infected with other sexually transmitted pathogens, which can amplify mucosal inflammation, leukocyte activation, and oxidative stress [22]. This was confirmed by the higher frequency of positive semen culture among *T. vaginalis*–positive men of this study. Such finding may reflect a common biological response that predispose certain men to multiple genital infections. Due to the retrospective design of the study and our interest on *T. vaginalis* PCR status, we cannot fully disentangle whether semen culture positivity is a mediator or a common feature for a broader inflammatory phenotype. Nonetheless, the reported co-infections with other STIs highlights that *T. vaginalis* early detection among infertile men is important and necessitates careful fertility evaluation for those individuals.

5. Conclusion

Our findings showed that *T. vaginalis* infection is associated with reduced semen quality, higher post-inflammation markers, higher oxidative stress markers in semen, and increased rates of DNA fragmentation. Providing a molecular method for detecting *T. vaginalis* in every infertility assessment procedure helps in providing information regarding a potentially curable disease that if treated can significantly improve the male fertilizing ability.

Article Information

Author contributions:
Abbas Ghafil Abbas: Carried out the study design, patients' selection, samples analysis, writing of the manuscript, revision and approving the final version.
Alaa Hachem: Carried out the design and implementation of the study, data collection and statistical analysis, writing of the manuscript, revision and approving the final version.

Ethics Approval: The study protocol was approved by the ethical committee at the College of Medicine- University of Al-Qadisiyah.

Data availability: All data are included in this article.

Conflicts of interest: The authors declare no conflicts of interest or financial interest to disclose.

Funding: No funding.

Acknowledgement: We extend our sincere appreciation to every participant in the study.

Disclaimer (Artificial Intelligence): The author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.), and text-to-image generators have been used during writing or editing of manuscripts.

Competing Interests: Authors have declared that no competing interests exist.

References

[1] W. D. Petok. *Procreation, Potency, and the Price of Paternity: The Social and Psychological Impact of a Diagnosis of Male Factor Infertility*. Urology, 2026.

[2] O. Fomichova, P. F. Oliveira, and R. L. Bernardino. Exploring the interplay between inflammation and male fertility. *The FEBS Journal*, 292(13):3321–3349, 2025.

[3] M. W. Russell, J. Whittum-Hudson, P. L. Fidel, E. W. Hook, and J. Mestecky. Immunity to Sexually Transmitted Infections. In J. Mestecky, W. Strober, M. W. Russell, B. L. Kelsall, H. Cheroutre, and B. N. Lambrecht, editors, *Mucosal Immunology*, chapter 11, pages 2183–2214. Academic Press, Boston, fourth edition, 2015.

[4] L. Tsoukalas, C. M. Vassalos, N. Gkitsakis, P. Gkotzamani, E. Gkoumalatsou, K. Bakalianou, et al. Trichomonas vaginalis in Vaginal Samples from Symptomatic Women in Greece: Assessment of Test Performance and Prevalence Rate, and Comparison with European Prevalence Estimates. *Acta Microbiologica Hellenica*, 70(3):29 pages, 2025. URL https://www.mdpi.com/2813-9054/70/3/29.

[5] S. A. Hosseini, M. Matini, M. Bahmanzadeh, R. Aslani, and F. Foroughi-Parvar. Trichomoniasis in Men: A Neglected Factor in Male Infertility? *Acta Parasitol*, 70(1):44, 2025.

[6] Z. Zhang, F. Li, Y. Deng, Y. Li, W. Sheng, X. Tian, et al. Trichomonas vaginalis excretory secretory proteins reduce semen quality and male fertility. *Acta Trop*, 238:106794, 2023.

[7] P. Sengupta, G. M. Pinggera, A. E. Calogero, and A. Agarwal. Oxidative stress affects sperm health and fertility-Time to apply facts learned at the bench to help the patient: Lessons for busy clinicians. *Reprod Med Biol*, 23(1):e12598, 2024.

[8] Y.-H. Gong, Y. Liu, P. Li, Z.-J. Zhu, Y. Hong, G.-H. Fu, et al. A nonobstructive azoospermic patient with Trichomonas vaginalis infection in testes. *Asian Journal of Andrology*, 20(1):97–8, 2018.

[9] WHO. *WHO Laboratory Manual for the Examination and Processing of Human Semen*. World Health Organization Press, Geneva, Switzerland, 2021.

[10] U. A. Jabr, H. A. M. Alsaady, and Z. S. Aziz. The Effect of Trichomonas vaginalis and some Urogenital Bacteria on Male Fertility in Maysan Province, Iraq. *Nigerian Journal of Parasitology*, 45(2):338–349, 2024.

[11] Z. Zhang, Y. Li, H. Lu, D. Li, R. Zhang, X. Xie, et al. A systematic review of the correlation between Trichomonas vaginalis infection and infertility. *Acta Trop*, 236:106693, 2022.

[12] K. Khalafalla, W. El Ansari, P. Sengupta, A. Majzoub, H. Elbardisi, O. Canguven, et al. Are sexually transmitted infections associated with male infertility? A systematic review and in-depth evaluation of the evidence and mechanisms of action of 11 pathogens. *Arab J Urol*, 21(4):216–232, 2023.

[13] R. Guiton and J. R. Drevet. Viruses, bacteria and parasites: infection of the male genital tract and fertility. *Basic and Clinical Andrology*, 33(1):19, 2023.

[14] I.-H. Han, J.-H. Kim, and J.-S. Ryu. Inflammatory response to Trichomonas vaginalis in the pathogenesis of prostatitis and benign prostatic hyperplasia. *PHD*, 61(1):2–14, 2023.

[15] J. Rosellen, F. Dittmar, A. Hauptmann, T. Diemer, H.-C. Schuppe, U. Schagdarsurengin, et al. Impaired Semen Quality in Patients with Chronic Prostatitis. *Journal of Clinical Medicine*, 13(10):2884, 2024.

[16] A. Potiris, E. Moustakli, E. Trismpioti, E. Drakaki, D. Mavrogianni, A. Matsas, et al. From Inflammation to Infertility: How Oxidative Stress and Infections Disrupt Male Reproductive Health. *Metabolites*, 15(4), 2025.

[17] E. A. El Saftawy, R. Y. Shash, H. M. Elwi, A. S. Gamal, M. F. Farag, B. E. Aboulhoda, et al. Impact of Escherichia coli, Candida non-albicans, and Trichomonas vaginalis on Semen Chemical and Functional Parameters: an In-Vitro Study. *Discov Med*, 36(184): 959–970, 2024.

[18] R. Henkel. Leukocytospermia and/or Bacteriospermia: Impact on Male Infertility. *J Clin Med*, 13(10), 2024.

[19] M. K. Panner Selvam, S. Baskaran, S. O'Connell, W. Almajed, W. J. G. Hellstrom, and S. C. Sikka. Association between Seminal Oxidation-Reduction Potential and Sperm DNA Fragmentation-A Meta-Analysis. *Antioxidants (Basel)*, 11(8), 2022.

[20] R. Henkel, U. Offor, and D. Fisher. The role of infections and leukocytes in male infertility. *Andrologia*, 53(1):e13743, 2021.

[21] H. Zhao, . C. Yu, C. He, C. Mei, A. Liao, and D. Huang. The Immune Characteristics of the Epididymis and the Immune Pathway of the Epididymitis Caused by Different Pathogens. *Front Immunol*, 11:2115, 2020.

[22] W. Tian, Y. Li, Y. Zhang, Y. Zhang, Y. Qin, Y. Han, et al. Systematic review and meta-analysis of the global prevalence and infection risk factors of Trichomonas vaginalis. *Parasite*, 32:56, 2025.