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Serological Investigation for *Trichomonas vaginalis* Infection and Oxidative Stress in Prostate Cancer Patients

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Abstract

Prostate cancer (PCa) is the most common cancer worldwide. Locally, PCa ranks fifth. Recent studies suggest a potential link between cancer development, oxidative stress, and persistent inflammation. The sexually transmitted parasite *Trichomonas vaginalis* has a clear pathogenic effect on females and has been linked to cervical cancer and reproductive problems in women. However, its impact on male diseases, particularly PCa, remains under investigation. This paper looked at the link between *T. vaginalis* infection and PCa, focusing on oxidative stress (Malondialdehyde, MDA) as a possible mechanism. A case-control research included 88 participants–58 PCa and 30 healthy controls from Babylon governorate, Iraq. ELISA was used to determine *T. vaginalis* seropositivity for IgG and IgM antibodies. Oxidative stress was assessed by measuring the MDA level. Statistical analysis comprised Kruskal-Wallis tests, Mann-Whitney U test, Fisher's exact test, and Chi-square. PCa patients showed much greater IgG seropositivity (25.86%) than controls (6.66%; p= 0.04). IgM revealed no notable correlation (p= 0.55). Although treatment type had no major impact on IgG status (p = 0.69), among cancer patients, IgG prevalence was highest in those receiving chemotherapy (34.48%). PCa patients showed significantly higher MDA levels (5.687 ± 3.276 nmol/mL) as oxidative stress marker than controls (2.056±1.890 nmol/mL; p<0.01). Patients on immunotherapy had the lowest MDA values (2.374±0.833 nmol/mL; but non-significantly p=0.097 compared radiotherapy/chemotherapy. In conclusion, PCa is associated with chronic *T. vaginalis* infection, which may be caused by oxidative stress or may be a triggering factor for the cancer.

Keywords: Malondialdehyde, Oxidative Stress, Prostate Cancer, *Trichomonas Vaginalis*.

Introduction

Prostate cancer (PCa) is one of the most widespread cancers globally. It is the fourth of 36 diagnosed types of cancer, at a rate of 7.3%, and the eighth in terms of causing deaths, at a rate of 4.1%. Its infections reached 1,466,680 cases and deaths reached 396,792 cases in 2022 (1). In recent years, the incidence of PCa has doubled significantly (13 times) and the death rate (9.5 times), while countries have undergone an increase in infections and deaths, such as African and Asian countries, Latin America, and Central and Eastern Europe. In contrast, other countries have witnessed a noticeable decrease in deaths from this disease. The high rates of infections and deaths in the first group of countries are due to an increase in diagnosis and a lack of access to treatment (2). In Iraq, PCa is among the most common cancers among men and ranks fifth in terms of prevalence, accounting for 7% for the years 2015-2018(3). The main causes of the cancer are many, but they are not explained precisely. They may have a genetic background that lies in defects or mutations in certain genes, or it is due to environmental factors, which constitute 50% of them, including infectious agents (4). Chronic inflammation brought on by infection seems to be the most important factor in PCa (5). One of the parasites that infect the human reproductive system is the flagellate parasite *T. vaginalis*, which is transmitted sexually between males and females (6). Data collected by the World Health Organization in 2021 indicate that the global prevalence of trichomoniasis reached 156 million new cases identified annually (7). Its pathogenic importance is clearly evident in females, causing vaginitis and some serious complications (8-10) and even cervical cancer (11, 12). Despite its moderate or mild pathogenicity in males (13), recent researches have confirmed its involvement in major problems and serious complications, including its association with prostate cancer (14-16). Only one study was found in Iraq linking *T. vaginalis* infection with prostate

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cancer, it was based on serological examination of IgG antibodies to the parasite, as well as investigation of its DNA. A positive relationship was identified between the appearance of IgG and prostate cancer, while the presence of the parasite's DNA in prostate tissue removed from patients was not recorded (17). Many hypotheses exist through which parasites can promote cancer or transform tissue, including inducing inflammatory reactions and stimulating the immune system, as some immune cellular mechanisms work to manipulate cellular signals, changing some metabolic pathways, which increases the chances of cancer occurring(18). Another important mechanism is that parasitic infections increase the production of free radicals and thus the occurrence of oxidative stress (19-21), which in turn is one of the important factors in the occurrence of cancer, as some free radicals combine with nitrogenous bases within the DNA structure, causing a defect in the genes responsible for some important diseases, including PCa (22). Investigating oxidative stress and serological indicators of *T. vaginalis* infection is essential for clarifying its possible involvement in the pathophysiology of prostate cancer. A chronic *T. vaginalis* infection triggers a sustained inflammatory response, producing reactive oxygen species (ROS) that inflict oxidative damage on host DNA and cellular components within the prostate microenvironment (23). This prolonged oxidative stress may foster carcinogenesis by enabling proto-oncogene activation and genomic instability (24). Consequently, particular serological indicators of prior infection yield critical epidemiological data, enabling researchers to associate exposure with oncogenic risk and investigate this mechanistically underexplored pathway (25). The relationship between *T. vaginalis* infection and PCa will be discussed in the current study, along with one of the key mechanisms underlying it and the use of MDA as a predictor of oxidative stress. This study attempts to elucidate a potential pathway mediated by oxidative stress, which may provide a mechanistic link missing in previous literature, providing new insights into prostate cancer prevention and highlighting oxidative pathways as potential targets for therapeutic intervention.

Methodology

Patients and Control

Eighty-eight [88] venous blood samples were gathered from men hospitalized at the Babylon Oncology Center from November 2024 to March 2025. The samples included 58 men diagnosed with PCa as confirmed cases were diagnosed by a professional physician who performed medical examinations on them working in oncology center, 30 healthy men without any diagnosed disease served as a control group. They were obtained from the same site and were mostly affiliated with the above-mentioned oncology center. The mean ages of participants were 67.97 and 62.03 years for patients and healthy controls, respectively. All participants, both patients and healthy individuals, were given a questionnaire that included the collection of the following data: name, age, duration of illness, type of treatment, marital status, presence of children, and whether the children suffer from any health issues.

Blood Samples Collection

Blood and serum samples were taken from all patients according to ethical guidelines that included some exceptions. The samples were collected under sterilization conditions using single-use medical syringes and medical gloves from Babylon Oncology Center. A 5 ml venous blood sample was collected from each participant under sterile conditions and kept in a gel tube to collect serum. The sample is placed in a centrifuge and spun at 3,000 rpm for 10 minutes. The upper layer, representing the serum, is withdrawn using a micropipette. It is then stored in Eppendorf tubes under freezing conditions at -20°C until use.

ELISA Assessment of Anti- *T. vaginalis* IgG and IgM Antibodies

The Human *T. vaginalis* IgG, TV IgG ELISA Kit Serial number Cat.NO ED0626Hu and the Human *T. vaginalis* IgM, TV IgM ELISA Kit Serial number Cat.NO ED0628Hu were used for qualitative diagnosis of the parasite's IgG and IgM antibodies. It was produced by BT Laboratory, China, and all steps were followed according to the manufacturer's manual.

Detection of Malondialdehyde levels

Human MDA ELISA Kit Serial number Cat.NO E1371Hu (BT Laboratory Co., China) were designed for the precise quantitative identification

of Human MDA in serum, plasma, Ascites, cell culture supernates, tissue homogenates and other biological fluids. All instructions in the enclosed kit user manual were followed and MDA levels in the samples were estimated according to the standard curve formed by reading the absorbance of prepared standard solutions of known concentrations.

Statistical Analysis

The statistical analysis was conducted using SPSS statistical software, version 26. Several tests were conducted according to nature of the data and the sample size, nonparametric tests were used, including chi-square for independence, Fisher's exact test, Mann–Whitney U and the Kruskal-Wallis tests, Post- hoc tests were also applied when necessary. The significance level for significant differences was adopted if the p values were greater than or equal to 0.05.

Results

Table 1 outlines several important factors related to the study participants for both groups (PCa patients and Healthy people), the study found statistically significant differences between those with and without PCa. Especially noteworthy was the age distribution's notable change (P = 0.016), which revealed a larger percentage of PCa patients in the 61–73 age range (50%) and a lower percentage in the 48–60 age range (18.97%). Treatment type now varied greatly (P = 0.000); among cancer patients, chemotherapy was the most frequent treatment (50%); among non-cancer people, no treatment was recorded. Cancer diagnosis (P = 0.001) was notably related to social status as all prostate cancer patients were married (100% vs. 83.33% in non-cancer people). With fewer surviving partners among cancer patients (77.58% vs. 96% in non-cancer people), partner status also shown relevance (P = 0.040). The cancer group also had more common children (P = 0.030; 94.82% vs. 80%). Disease onset length showed no notable variation, nevertheless (P = 0.270).

Table 1: The Study Group Characteristics

Variables	Total		Prostate Cancer Patients		Healthy People		*P* value Chi-square test
	n	%	n	%	n	%	
Total	**88**	**100**	**58**	**65.90**	**30**	**34.09**	
Age (years)	65.94 ± 9.40		67.97 ± 9.04		62.03 ± 8.97		
Age groups (years)							0.016
48–60	25	28.41	11	18.97	14	46.67	
61–73	41	46.59	29	50.00	12	40.00	
74–86	22	25.00	18	31.03	4	13.33	
Type of treatment							0.000
Chemotherapy			29	50			
Radiotherapy			7	12.06			
Hormonal therapy			18	31.03			
Immunotherapy			3	5.17			
Without treatment			1	1.72			
Onset of the disease (Time)							0.270
One year and less			20	34.48			
More than one to Two years			14	24.13			
More than two years			24	41.37			
Social status							0.001
Married	83	94.31	58	100	25	83.33	
Not married	5	5.68	0	0	5	16.66	
Partner status							0.040
Alive	69	83.13	45	77.58	24	96	
Dead	14	16.86	13	22.41	1	4	
Possessing offspring							0.030
Yes	79	89.77	55	94.82	24	80	
No	9	10.22	3	5.17	6	20	

T. vaginalis infection was diagnosed serologically by detecting IgM and IgG antibodies to the parasite using ELISA technique in two groups of PCa patients and healthy controls. The results are presented in Table. 2, which shows a significant difference ($P < 0.05$) in the seroprevalence of IgG antibodies between the two groups, where the probability value was 0.044, that 15 patients showed seropositivity for IgG immunoglobulin at a rate of 25.86%, while the prevalence of IgG in the control group was only two cases at a rate of 6.66%.

Table 2: Seroprevalence of *T. vaginalis* - IgG in the Study Groups

Group Study		IgG	
		Negative	Positive
healthy	Count	28	2
	%	93.33	6.66
Patients (Prostate cancer)	Count	43	15
	%	74.14	25.86
Total	Count	71	17
	%	80.68	19.32
P=0.044, Fisher's Exact Test			

Table 3: Seroprevalence of *T. vaginalis* -IgM in the Study Groups

Group Study		IgM	
		Negative	Positive
healthy	Count	30	0
	%	100	0
Patients (Prostate cancer)	Count	55	3
	%	94.83	5.17
Total	Count	85	3
	%	96.59	3.41
P = 0.548, Fisher's exact test			

The result in Table 3, showed that there was no significant difference ($P > 0.05$) in the presence of IgM antibodies between the two groups, as the probability value was 0.548 at using Fisher's exact test. The higher percentage of IgM positivity in PCa patients (5.17%) compared to healthy individuals (0%) means that there is no relationship between the current infection with the parasite and PCa. In PCa patients, therapy type had no appreciable correlation with *T. vaginalis* -IgG status (p=0.685, Fisher's exact test), However, the highest serological prevalence of IgG was in patients undergoing chemotherapy (34.48%) showed in Table 4.

Table 4: Seroprevalence of *T. vaginalis* - IgG in the Prostate Cancer Patients According to the type of Treatment Used

Type of Treatment	*Trichomonas* IgG				Total	
	Positive		Negative			
	n	%	n	%	n	%
Chemotherapy	10	34.48	19	65.52	29	50
Radiotherapy	1	14.29	6	85.71	7	12.1
Hormonal therapy	4	26.67	14	77.78	18	31.03
Immunotherapy	0	0	3	100	3	5.17
Without treatment	0	0	1	100	1	1.72
Total	15	25.86	43	74.14	58	100
P=0.685, Fisher's exact test						

Table 5: Concentrations of MDA in the Sera of Healthy and Prostate Cancer Groups

Study groups	N	Mean nmol/ml	Std. Deviation	Std. Error
HEALTHY	30	2.056	1.890	0.345
PROSTATE CANCER	58	5.687	3.276	0.430
Total	88	4.449	3.350	0.357

The results of measuring MDA levels in the sera of PCa patients and healthy controls are shown in Table 5, there is a significant increase P < 0.01 (Mann-Whitney U test) in the level of MDA in PCa patients (5.687± 3.276 nmol/ml), compared with the control group (2.056± 1.890 nmol/ml). The Kruskal-Wallis test revealed significant differences in mean MDA levels between the study groups (H=32.033, df=3, p<0.001). Post hoc comparisons (Mann-Whitney U) indicate that Healthy individuals had significantly lower MDA levels compared to the PCa (cancer with and without parasite infection) groups, Table 6.

Table 6: Concentrations of MDA in the Sera of the Study Groups

Study groups	N	Mean	Std. Deviation	Std. Error
Healthy	28	1.966[a]	1.905	0.360
Prostate cancer	43	5.677[b]	3.416	0.521
Prostate cancer with trichomonas	15	5.716[b]	2.953	0.763
Trichomonas only	2	3.312[ab]	1.489	1.053
Total	88	4.449	3.350	0.357

Different alphabetical letters between groups indicate significant differences at the 0.05 level

Table 7: Malondialdehyde Levels in Prostate Cancer Patients according to Treatment Type

Type of treatment	N	Mean	Std. Deviation	Std. Error
Chemotherapy	29	6.241	3.477	0.646
Radiotherapy	7	7.279	3.610	1.365
Hormonal therapy	18	4.837	2.629	0.620
Immunotherapy	3	2.374	0.833	0.481
Total	57	5.722	3.295	0.437

Table 7 shows that by applying the Kruskal-Wallis test non-significant differences in mean MDA levels between treatment groups (H=6.318, df=3, p=0.097). However, Immunotherapy has lower MDA levels compared to Radiotherapy, Chemotherapy and Hormonal therapy.

Discussion

More than 170 million instances of trichomoniasis every year are caused by the parasite protozoan *T. vaginalis*, making it the most common sexually transmitted illness, because of its diverse appearance and problems with diagnostic testing, the infection can be difficult to identify (26). Several methods have been used to diagnose *T. vaginalis* infection (27-29), and despite the positive aspects of each method, they are all fraught with drawbacks. Most diagnostic methods shed light on infections in females, but they remain limited for diagnosing infections in males (30). One of these methods, the serological technique, can offer a quick, accurate, and cost-effective approach to investigating the epidemiology of *T. vaginalis* infection (31).

The results of Table 2 show a much greater seroprevalence of *T. vaginalis* IgG antibodies in PCa patients than in healthy people, indicating a possible link between *T. vaginalis* infection and PCa. These findings are consistent with other researches, a multi-country meta-analysis study found that *T. vaginalis* infection is associated with human reproductive cancers, and infection with this parasite can lead to cervical cancer or PCa (18). Another study suggested that *T. vaginalis* seropositivity was associated with a higher PCa risk by postulating chronic inflammation as a contributory factor (25). In the same vein, researchers discovered a favourable serological link, hence confirming the involvement of infectious pathogens in cancer development (32). A local study demonstrated a significant association between IgG antibodies to *T. vaginalis* and PCa (OR = 3.895, p = 0.047), suggesting that chronic infection can increase the risk of cancer via inflammation, although direct *T. vaginalis* DNA was not found in prostate tissue (17). Contradictory results indicate no meaningful association, draw attention to methodological variation in serological tests or demographic diversity as possible confounding factors (33). Our current results are also inconsistent with the findings of a nested case-control study that demonstrated that

T. vaginalis seropositivity is not associated with PCa in Caucasian or African American men (34). A previous study also showed no significant relationship between PCa and the presence of Trichomonas antigens (35).

Several studies have proposed hypotheses about the different mechanisms underlying cancer induction by *T. vaginalis* infection. One mechanism has shown that the parasite *T. vaginalis* produces small vesicles containing various chemical substances that combine with the host cells and excrete their contents inside, leading to modulation of the immune response and increased parasite adhesion to the host cells, especially prostate gland cells (36). Other researchers have put forward a testable hypothesis, supported by evidence, that *T. vaginalis* stimulates PCa by producing IL-6, which leads to up regulation of a signalling cascade responsible for modifying the activity of certain tumour-initiating genes (37). In a more recent study than the two previous studies, it was found that *T. vaginalis* secretes a protein called Macrophage migration inhibitory factor (MIF), a parasite-specific protein which is 47% similar to the human type, the study demonstrated that this factor has a direct effect on the growth of PCa tissue outside the living body, confirming the parasite's role in inducing inflammation and stimulating tissue growth in the emergence of PCa (23). On the other hand, the same parasite causes intracellular, mitochondrial ROS and ER generation in SiHa cells, leading to DNA damage and ultimately apoptosis by several cellular pathways (21, 38).

The current results indicate non-significant P-value 0.685 points to no obvious link about treatment type effects on the seroprevalence of *T. vaginalis* IgG antibodies in PCa patients, though treatment kind and serological status seem to differ obviously; those getting chemotherapy had the highest positivity rate (34.48%), followed by hormonal therapy (26.67%) and radiotherapy (14.29%). Patients undergoing immunotherapy or those without treatment showed no positive cases; however, sample sizes in these groups were small (3 and 1, respectively). Our findings are supported by a previous study which suggested that immunotherapy can reduce persistent infections by improving immune responses (39). On the other hand, immunosuppression caused by chemotherapy may prolong the presence of infection-associated antibodies, and thus this may be the reason for the greater prevalence in this population (40). The incidence of chronic *T. vaginalis* infection in patients receiving hormone therapy was relatively high (26.67%). The effect of hormone therapy (ADT) on chronic *T. vaginalis* infection in men is unclear and requires further research. Potential effects may range from environmental changes that promote persistent infection to decreased clearance due to altered immunity. Scientific studies demonstrate that ADT modifies the prostate microenvironment, so lowering inflammation first but maybe causing atrophic alterations and changed local immunity with time (41, 42). This might possibly affect the elimination or persistence of *T. vaginalis*. ADT has been shown to cause prostate atrophy and notable variations in prostatic fluid composition (low zinc levels). Although this is speculative and has not been specifically proven for *T. vaginalis*, zinc has antimicrobial properties, and its lowering would potentially produce a less hostile environment for *T. vaginalis* survival (43).

The present study found a significant increase ($P < 0.01$, Mann-Whitney U test) in serum MDA levels in PCa patients (mean: 5.687 nmol/ml) relative to healthy controls (mean: 2.056 nmol/ml), hence highlighting oxidative stress as a possible cause of PCa. These results are consistent with a previous study that showed higher MDA levels in prostate cancer patients (mean: 5.2 nmol/ml) and linked lipid peroxidation to tumor progression (44). In past study demonstrated that MDA is a measure of oxidative DNA damage in PCa (45). Our results are also aligned with those of another study, although the MDA level in the current study was higher than in that study (46), which may be due to differences in testing techniques or disease stage. Elevated MDA levels in patient samples indicate systemic oxidative imbalance, MDA has previously been implicated in carcinogenesis resulting from chronic inflammation (47). MDA levels may be affected by other confounding factors such as comorbidities, antioxidant therapy, etc. (48). The present study demonstrated significantly elevated serum MDA in PCa patients and those with PCa plus *T. vaginalis* IgG compared to healthy controls, emphasizing oxidative stress as a hallmark of prostate carcinogenesis. This is supported by other researchers who identified MDA as a key mediator of tumor aggressiveness (44). Another study also

linked lipid peroxidation to DNA damage in malignant tumors (45). Notably, the modest MDA elevation in the Trichomonas IgG-only group suggests infection alone may induce mild oxidative stress, nevertheless limited by a small sample size (n = 2), It has been previously reported that chronic *T. vaginalis* infection exacerbates inflammation (25). These results demonstrate the interaction between infection and oxidative stress in PCa, as previously hypothesized by another study (32).

The study found non-significant variations in MDA levels among PCa treatment groups (P = 0.097, Kruskal-Wallis test), though immunotherapy showed pointedly lower MDA levels (mean: 2.374 nmol/mL) than chemotherapy (6.241 nmol/mL), radiotherapy (7.279 nmol/mL), and hormonal therapy (4.837 nmol/mL). The results regarding the levels of MDA in patients receiving immunotherapy do not agree with a past study, which indicated that cancer immunotherapies can alter tumor oxidative status, as it leads to increase tumor oxidative stress (49). This discrepancy may be due to the small sample size (n = 3) of those receiving immunotherapy in the current study. On the other hand, the increased MDA in the chemotherapy and radiation groups is explained by a previous study that confirmed how cytotoxic treatments increase ROS generation, thus intensifying oxidative damage (50). The mean MDA levels for hormone therapy are consistent with what other researchers have observed, as they noted significantly lower levels and activity of antioxidant enzymes in erythrocytes, while plasma MDA levels were higher in PCa patients compared to the healthy group. With anti-androgen treatment, there was a significant decrease in plasma MDA levels in the patient group (51). MDA levels were found to be elevated in PCa patients who had not undergone treatment, and this compound remained elevated in patients undergoing hormonal therapy compared to the control group, but its levels decreased slightly compared to patients who had not undergone hormonal therapy (52).

Conclusion

The current study provides the first experimental evidence in a specific population that links *T. vaginalis* infection with oxidative stress, a biological pathway known to induce carcinogenesis. The case-control current study found a notable link between chronic *T. vaginalis* infection (indicated by raised IgG seropositivity: 25.86% in PCa patients vs. 6.66% in controls) and PCa, possibly mediated by oxidative stress, as shown by higher MDA levels in patients. Although IgM had no relationship with active infection, chronic inflammation and oxidative damage were reasonable explanations connecting *T. vaginalis* to cancer creation. Immunotherapy linked to lower MDA levels, indicating oxidative stress pathways as treatment targets. These results highlight the need for screening high-risk groups for *T. vaginalis* and investigating antioxidant-based treatments to slow the progression of PCa. This study tests the hypothesis that chronic *T. vaginalis* infection contributes to PCa pathogenesis by causing a state of persistent oxidative stress that results in macromolecular damage and genomic instability. It does this by combining seroprevalence data with direct quantification of oxidative stress within the same cohort of PCa patients and controls. Additionally, it produces important population-specific data from an understudied Babylonian, Iraqi population, where the high prevalence of PCa and possibly a unique STIs profile may produce results that differ from those of Western cohorts that have been previously studied.

Limitations of Study

The reasons for the study's limitations included the practical and ethical challenges of collecting tissue samples, the difficulty of recruiting patients from a single location, a tight budget, and a deadline for submitting the dissertation. These are all examples of the realities of clinical research. Within these constraints, we planned our study to provide an effective answer to our research question.

Abbreviations

ADT: Androgen deprivation therapy, DNA: Deoxyribonucleic Acid, ELISA: Enzyme-Linked Immunosorbent Assay, ER: Endoplasmic Reticulum, IgG: Immunoglobulin G, IL: Interleukin, IgM: Immunoglobulin M, MDA: Malondialdehyde, MIF: Macrophage migration inhibitory factor, PCa: Prostate cancer, ROS: Reactive Oxygen Species, SD: Standard deviation, SiHa: Sarcoma invasive Harris, SPSS: Statistical Package for the Social Sciences, STIs: Sexually Transmitted Infections.

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Author Contributions

Zahraa R. Atiyah: all the work, writing, Raad A. Kadhim: supervision, Ali H. Al-Marzoqi: supervision.

Conflict of Interest

The authors declare no conflict of interest.

Declaration of Artificial Intelligence (AI) Assistance

The authors declare no use of artificial Intelligence (AI) for the write up of the manuscript.

Ethics Approval

Ethical permission was granted by the Research Ethics Committee at the College of Science for Women/University of Babylon in the form developed for this purpose and numbered 32 on 28/6/2024, based on meeting the requirements and consent of patients and research participants.

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