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Original Research Article

The Relationship between Laten Toxoplasmosis and Infertility Parameters of Men in Basra Province-Iraq

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Abstract: ***Background*:** Toxoplasmosis is caused by the obligatory intracellular parasite *T. gondii*, about one-third of the world is afflicted with this disease, which is among the most prevalent in the globe. ***Objective***: Establish the seroprevalence of the *Toxoplasma gondii* infection in fertile and infertile men in the Basra province, Iraq, and to determine the relationship between chronic infection with *T. gondii* and semen parameters and reproductive hormone levels. ***Methodology*:** A cross-sectional study was carried out on 214 fertile and infertile men in Basra, semen and blood samples were taken, the Latex Agglutination Test was used to check the presence of *T. gondii* antibodies. Serum concentration of FSH, LH, prolactin, and testosterone was measured by using automated immunoassay system. ***Results***: The general rate of IgG seroprevalence against *T. gondii* of the participants was 23.8% and it was marginally higher in infertile men (27%) compared to fertile men (21%), but with not statistically significant (p=0.39). IgM was infrequently detected and was found only in one infertile individual. Rapid progressive motility was lower significantly among IgG-positive people among fertile men (p=0.034). Hormonal analysis revealed no significant differences in FSH, LH, prolactin, or testosterone levels between infected and non-infected individuals. ***Conclusion*:** The latent *T. gondii* infection is quite frequent in the Basrah province in men, but it does not have a strong correlation with infertility of men as a whole or significant hormonal disruptions. The chronic toxoplasmosis can have a weak functional impact on the quality of sperm as opposed to a quantitative or endocrine one.

Keywords: Latentoxoplasmosis, Infertility, Hormones, Semen, Basra province.

INTRODUCTION

Toxoplasmosis is caused by the obligatory intracellular parasite *T. gondii*, about one-third of the world is afflicted with this disease, which is among the most prevalent in the globe [1]. *T. gondii* has a diverse range of hosts and a complicated life cycle, the only known definitive hosts of *T. gondii* are felids, both domestic and wild, even though practically all warm-blooded animals can become infected.

There are both sexual and asexual cycles, but only the asexual cycle happens in intermediate hosts, such as humans and animals [2]. *T. gondii* is transmitted to humans in several ways depending on the type of stage, the most common way is through food by eating raw or undercooked meat that has tissue cysts, sporulated *T. gondii* oocysts, in the fecal of infected cats in the environment is a possible source of infection, and ingesting unwashed raw vegetables or fruits [3], from acutely infected pregnant women to embryo by placenta and receiving an organ donation are all ways that *T. gondii* is spread [4, 5].

In a systematic review, it was found that *Toxoplasma* is typically found in various organs including the male and female reproductive organs of intermediate hosts, following parasite ingestion and tachyzoite proliferation during the acute stage of its life cycle, as result the infection may have some effects on the reproduction process [6]. Male infertility problems have multiple causes; toxoplasmosis may be one of them, but the mechanisms of this phenomenon are not clear yet. Studies on laboratory animals, especially mice, have shown a negative impact on sperm and endocrine

function. It has also been proven that the parasite may be sexually transmitted, as it has been found in the seminal fluid in humans [7]. The sexual transmission of the parasite remains hypothetical due to limitations in research; therefore, further studies are needed to determine the impact of toxoplasmosis infection on the male reproductive system.

METHODOLOGY

Region of Study

The study was carried out in Basra province, blood samples and seminal fluid were collected from fertile and infertile man at Dar alshefaa investment hospital during November 2023 to March 2024, a questionnaire was designed to ensure that they were free of hereditary diseases and record the age, region, and date of sample collection.

Samples Collection

Blood Collection

A total of 214 venous blood samples and seminal fluid were collected from fertile and infertile man, five ml of venous blood was drawn into gel tube for serum separation and left for 10 minutes then transported into centrifuge which was fixed on 3000 rpm for about 5 minutes, centrifuged sera were separated from gel tubes. The serum divided in two plain tubes, one tube for toxoplasmosis test and other for hormone tests.

Semen Collection

After a period of at least 3 days of sexual abstinence, the semen samples of the patients were scrutinized and they were all given a clean, wide mouth, sterile, dry, graduated, plastic and warm disposable container. The container was put with the name of the man, his identification number and time and date of collection, the seminal fluid samples were immediately incubated at 37 C and were left to completely liquid (30 minutes).

Serodiagnosis of Toxoplasmosis

***Latex Agglutination Test*:** The Procedure was done according to the manufacturing instructions (Salucea company)

***Toxo IgG/IgM Combo Rabid Test*:** The positive samples were conduct also by Toxo IgG/IgM Combo Rabid Test according to the manufacturing instructions.

Semen Analysis

Following the liquefaction of the sample, sperms motility and morphology, were evaluated using WHO standard procedures for *Toxoplasma* seropositive and 25 of seronegative as control.

Sperms Motility

A representative amount of semen (10 µl) was dropped onto a glass slide that was covered with phase-contrast optics under the magnification of x 200 or x 400 and examined. They sperms motility was affected by abnormality in men whose motility was progressive (PR) less than 32%.

Hormones Analysis

A total of (51 were positive for *Toxoplasma* and 25 were negative as control) were tested for LH, FSH, Prolactin and Testosterone hormones by COBAS e 411 device.

Statistical Analysis

The SPSS (Ver. 25) program was used in this study for the purpose of statistical analysis of data which represented by using the Chi square, each analysis was made under probability level $p \leq 0.05$.

RESULTS

Out of 214 male participants, 51 (23.8%) tested positive for anti-*T.gondii* IgG antibodies, indicating past exposure to the parasite. Among them, 27 out of 100 (27%) were from the infertile group, while 24 out of 114 (21%) were from the fertile group. A Chi-square test showed no statistically significant difference in IgG seropositivity between fertile and infertile males (p=0.39), suggesting that previous *Toxoplasma* infection is not significantly associated with male infertility in this study population. As for acute infection, only one participant (1%) from the infertile group tested positive for anti-*Toxoplasma* IgM antibodies, while none of the fertile participants tested positive. The difference in IgM positivity between the two groups was also not statistically significant (p = 0.948), indicating that active or recent infection was not common among the participants (Table 1).

Table 1: Seroprevalence of *T. gondii* antibodies among fertile and infertile males

Groups	IgG				IgM		
	n	**+ve toxo**	**%**	**p-value**	**+ve toxo**	**%**	**p-value**
Fertile	114	24	21.0	0.39	0	0	0.94
Infertile	100	27	27		1	100	
Total	**214**	**51**	**23.8**		**1**	**0.46**	

The comparison of semen parameters between *Toxoplasma*-positive (Toxo.+ve) and *Toxoplasma*-negative (Toxo.-ve) individuals within both infertile and fertile groups is presented in the (Table 2). For clarity, abbreviations used in the table are explained in the footnote. In the infertile group, there were no statistically significant differences between Toxo. +ve and Toxo. -ve individuals in semen volume (p=0.956), sperm concentration (p = 0.310), total sperm count per ejaculation (p=0.397), or rapid linear progressive

motility (p= 0.759). However, significant differences were observed in slow or sluggish progressive motility, which was higher in Toxo. +ve individuals (p = 0.003), non-progressive motility, also significantly higher among Toxo. +ve individuals (p = 0.000). Additionally, non-motile sperm showed a marginal significance (p=0.066), indicating a possible trend toward reduced motility in infected individuals. In the fertile group, semen volume, sperm count, and most motility parameters showed no statistically significant differences between infected and non-infected individuals. However, a significant reduction in rapid progressive motility was observed in Toxo. +ve individuals (p=0.034), suggesting that even in fertile men, *T. gondii* infection may negatively impact sperm motility.

Table 2: Comparison of SFA parameters in *T. gondii* IgG positive and *T. gondii* IgG negative among infertile and fertile men

Parameters	Group	Infertile Mean	Sig.	Fertile Mean	Sig.
Vol. (ml)	Toxo +ve	2.589	.956	3.60	.058
	Toxo -ve	2.607		2.969	
Conc. ($\times 10^6$/ml)	Toxo +ve	17.96	.310	68.46	.497
	Toxo -ve	13.24		65.20	
Tot. count/Ejac.	Toxo +ve	53.41	.397	228.2	.109
	Toxo -ve	35.69		193.5	
Rapid prog. motility	Toxo +ve	3.18	.759	7.29	.034
	Toxo -ve	2.71		10.67	
Sluggish prog. motility	Toxo +ve	12.86	.003	23.96	.214
	Toxo -ve	7.50		21.67	
Non-prog. motility	Toxo +ve	27.43	.000	25.92	.787
	Toxo -ve	12.78		25.08	
Non-motile sperms	Toxo +ve	52.96	.066	42.83	.787
	Toxo -ve	43.26		42.33	

Abbreviations: Vol. = volume; Conc. = concentration; Tot. count/Ejac. = total sperm count per ejaculation; Rapid prog. motility = rapid progressive motility; Sluggish prog. motility = slow or sluggish progressive motility; Non-prog. motility = non-progressive motility; Non-motile sperms = immotile spermatozoa.

(Table 3) showing the hormonal levels of infertile and fertile males according to their *T. gondii* infection status. For FSH, infertile males showed nearly identical mean levels between infected (5.34) and non-infected (5.27) subjects, with no statistical significance (p = 0.965). Similarly, fertile males displayed comparable levels between infected (4.30) and non-infected (3.74) individuals (p = 0.521).

In the case of prolactin (PRO), infertile males with *T. gondii* infection demonstrated a higher mean value (18.0) than non-infected individuals (13.3), but the difference was not significant (p = 0.441). In fertile males, there was a higher level of prolactin in non-infected males (19.49) than in infected males (14.20), but not significantly different (p = 0.103). In the testosterone (TEST) levels, there was a low difference in the mean concentration between the infected infertile males (5.47) and the non-infected (3.22) although the difference was not significant (p = 0.214). in fertile males the mean testosterone level of the infected males (5.89) was greater than that of their non-infected (4.32) male with no significant association (p = 0.102). In luteinizing hormone (LH) there were no significant differences, the mean of infertile infected males was 5.62 as opposed to non-infected subjects (5.44) (p = 0.896). Among fertile males, non-infected individuals had slightly higher LH levels (6.09) relative to infected ones (5.29), with no significant association (p = 0.275).

Table 3: Comparison of hormonal profiles (FSH, Prolactin, Testosterone, and LH) in fertile and infertile males according to *T. gondii* infection status

Hormone	Infertile			Fertile	
	Group	Mean	Sig.	Mean	Sig.
FSH	Toxo. +ve	5.34	.965	4.30	.521
	Toxo. -ve	5.27		3.74	
PRO	Toxo. +ve	18.0	.441	14.20	.103
	Toxo. -ve	13.3		19.49	
TEST	Toxo. +ve	5.47	.214	5.89	.102
	Toxo. -ve	3.22		4.32	
LH	Toxo. +ve	5.62	.896	5.29	.275
	Toxo. -ve	5.44		6.09	

(Table 4) presents the prevalence of *T. gondii* infection among men with different semen characteristics. The infection rates varied across groups, being 11% in azoospermia men, 17% in normozoospermia men, 24% in asthenozoospermic men, and the highest rate of 34% in oligozoospermic men. Although there was a clear trend of higher infection prevalence in men with reduced sperm quality, but these differences not statistically significant (p = 0.110). This suggests that, while *T. gondii* infection appears more frequent in oligozoospermic and asthenozoospermic individuals, the association between infection status and semen parameters cannot be confirmed with statistical confidence in this dataset.

Table 4: Seroprevalence of *T. gondii* antibodies in groups of men according to the number of spermatozoids

Number of spermatozoids	n	+ve toxo	%	Sig.
Azoospermia	27	3	11	0.11
Normozoospermia	27	5	17	
Asthenozoospermia	66	16	24	
Oligozoospermia (< 15 million/ml	52	18	34	

DISCUSSION

The aim of the current study was to investigate the chronic infection of toxoplasmosis in infertile and fertile men in Basra-Iraq, the overall seroprevalence of *T. gondii* IgG antibodies among men was 23.8%, with slightly higher rates in infertile (27%) compared to fertile (21%) males, though the difference was not statistically significant (p = 0.39). IgM positivity was rare, observed in only one infertile participant (0.46% overall). These findings indicate that chronic infection is relatively common, while recent or acute infection is infrequent in this population. We find our findings to agree with what is being reported in other areas, which documented a slightly higher rate of 25% from immunocompetent male in Iran [8]. These similarities point to a uniformity in moderate levels of IgG and very low levels of IgM in the diverse male subsets. Similarly, these inconsistencies could be due to variation in geographic exposure, diagnostic testing, or a choice in clinical versus community-based subjects. In general, our results confirm the prevalence of latent toxoplasmosis among men (20-30%) but not recent infection.

The current research indicated that the effect of *T. gondii* seropositivity was significant on the parameters of sperm motility among infertile men, as the parameters of sluggish progressive and non-progressive motility were higher in seropositive than seronegative men, but the other parameters like semen volume, sperm concentration and total number of sperms per ejaculation were not significantly different between groups. Only the rapid linear progressive motility was significantly decreased in *T. gondii*-positive individuals in fertile men only, which is compatible with the fact that the influence of the parasite is weak in fertile populations, but stronger in infertile ones.

T. gondii tachyzoites invade and develop in the testes and epididymis, disrupting the homeostasis of tissue structure, and leading to the immune cell infiltration and cell destruction. Along with proving that *T. gondii* is infective in the testes and epididymis, in vitro experiments showed a direct contact between *T. gondii* tachyzoites and human spermatozoa. This left a large percentage of headless spermatozoa [9]. On the other hand, there is no significant relationship between *T. gondii* seropositivity and semen quality, such differences can be explained by the variation of study populations, sample size, diagnostic methods, and host immune responses [8].

The current evidence, in general, suggests that the impact of chronic *T. gondii* infection on male fertility is more functional rather than quantitative, instead of decreasing the number of sperm or the amount of ejaculate, the parasite mainly alters flagellar patterns, which could be through mitochondrial dysfunction of spermatozoa, structural damage to flagella, or immunological changes in seminal, this may be the reason as to why seropositive fertile men can still retain reproductive competence despite minor defects in motility [10]. In this study, there were no significant differences in the FSH, LH, prolactin, or testosterone levels in seropositive and seronegative men with *T. gondii* in their fertile and infertile groups. Even though the testosterone levels were found to be slightly higher in seropositive men, the difference was not found to be statistically significant. This indicates that there is no significant effect of the pituitary-gonadal axis in chronic toxoplasmosis. Our results are consistent with other studies which found that in men, most of the studies had no difference in testosterone levels in relation to latent toxoplasmosis or only a slight increase11]. It was also emphasized that alterations tend to be observed in the ratio of testosterone to estradiol, as opposed to testosterone, it affects hormonal balance, but it is not a major endocrine disorder [12].

On the other hand, another study was found greater FSH and LH and lower testosterone in infected men in Iraq [13], which is contrary to our findings, these discrepancies can be associated with the disparity in study populations, stage of infection or laboratory procedures. Experimental evidence also supports the possibility that *T. gondii* can directly affect Leydig cells and interfere with testosterone production, even if systemic hormonal changes are not always detectable [14]. So, the present results indicate that *T. gondii* infection in men has, at most, a mild and variable influence on reproductive hormones, suggesting that its

role in infertility is more likely linked to local effects on sperm function than to major hormonal alterations.

In this study, the seroprevalence of *T. gondii* antibodies was higher among men with oligozoospermia (34%) and asthenozoospermia (24%) compared to those with normozoospermia (17%) or azoospermia (11%), although the differences did not reach statistical significance ($p = 0.11$). This trend suggests that *T. gondii* infection may be more common in men with reduced sperm count and motility. Similar findings were reported that latent toxoplasmosis in men was associated with lower sperm concentration and progressive motility [15]. Experimental data also confirm this correlation because *T. gondii* has been demonstrated to affect sperm mitochondrial activity and elevate DNA fragmentation, which may be the sources of low count and motility [14]. These results suggest that chronic toxoplasmosis can affect impaired semen quality, at least sperm concentration and motility, although not necessarily significantly.

CONCLUSIONS

This paper shows that latent *T. gondii* infection is quite frequent in the Basra province in men, but it does not have a strong correlation with infertility of men as a whole or significant hormonal disruptions. Though no major differences were identified in the sperm concentration, sperm volume, or hormone values, there were evident impairments in the parameters of sperm motility in infected men in general and infertile men in particular. These results indicate that chronic toxoplasmosis can have a weak functional impact on the quality of sperm as opposed to a quantitative or endocrine one. It is also advisable that further massive research be conducted to shed more light on the biological pathways through which *T. gondii* infection is associated with male reproductive performance.

Ethical Approval

Ethical approval was obtained from the Ethics Committee of Dar Al-Shifa Hospital, Basra, Iraq (Approval No. DSH/063/2024). Written informed consent was obtained from all participants. The study was conducted in accordance with the Declaration of Helsinki and relevant ethical guidelines.

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