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Original Article

Effect of *Toxoplasma gondii* Infection on the Level of Luteinizing and Follicle Stimulating Hormones in Women with Polycystic Ovary Syndrome

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Abstract

Background: The protozoan parasite *Toxoplasma gondii* is responsible for the most significant zoonotic illness in the world, Toxoplasmosis. It is possible for *T. gondii* to be transferred transplacentally from the mother to the fetus if it is contracted during pregnancy and causes abortion or congenital deformity. **Objective:** To investigate the correlation between polycystic ovarian syndrome and toxoplasmosis, as well as to examine certain related sex hormones, luteinizing hormone (LH) and follicle stimulating hormone (FSH). **Materials and Methods:** The current study was conducted at Kirkuk General Hospital and Gynaecological and Pediatric Hospital in Kirkuk city, Iraq, between November 1, 2022 and March 21, 2023. The study included 200 blood samples collected from women who had abortions. The serum used to measure levels of LH and FSH. **Results:** Out of the 200 aborted women, only 40 (20%) of them were PCO positive (poly cystic ovarian syndrome). The 40 positive PCO subjects were divided into four groups according to the presence of *T. gondii* antibodies: PCO +ve and Toxo IgM +ve (7.5%), PCO +ve and Toxo IgG +ve (15%), PCO +ve and Toxo IgM -ve (42.5%), PCO +ve and Toxo IgG -ve (35%). **Conclusion:** There were significant differences in the FSH and LH level in the study groups. The level of LH in four groups the highest mean was in Toxo IgM and PCO +ve (9.190 ± 3.710) with *P* value 0.046. In the level of FSH in four groups the highest mean was in Toxo IgG +ve and PCO +ve (8.776 ± 2.061) and the lowest mean was in Toxo IgM +ve and PCO +ve (5.790 ± 3.25) with a *P* value of 0.045.

Keywords: Follicle stimulating hormone, luteinizing hormone, polycystic ovarian syndromes, *Toxoplasma gondii*

Introduction

The parasite *Toxoplasma gondii*, is the primary cause of the zoonotic disease toxoplasmosis, which is present across the world.[1] It is a significant tropical disease that is expanding globally and is infectious to 30% of people worldwide.[2] *Toxoplasma gondii* has three infectious forms, including sporozoites found in oocysts, tachyzoites, and bradyzoites that reside in tissue cyst.[3] Eating raw or undercooked meat from other animals and carrying tissue cysts harboring bradyzoites are two ways that *T. gondii* can be transmitted to human hosts. After intermediate hosts consume the oocytes, the parasite excysts invade the enterocytes and cells of the ileal epithelium, and then transform into the rapidly replicating stage known as tachyzoites. Through a process known as endodyogeny, tachyzoites can enter the majority of nucleated host cells, where they can then form a parasitophorous vacuole and reproduce. The parasite will continue to divide inside the host cell until the host cell bursts, releasing the tachyzoites.[4] Among the numerous methods, including serological, histological, molecular, the enzyme linked immunosorbent assay is the more popular and reliable serological test.[5] The most common hormonal problem in women of childbearing age is polycystic ovarian syndrome (PCOS), which is linked to infertility. Both environmental and genetic variables are involved in the syndrome's

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etiology. There have been theories about polycystic ovarian disease, abnormalities of the hypothalamic-pituitary-ovarian or adrenal axis. A relative increase in the release of luteinizing hormone (LH) to follicle stimulating hormone (FSH) results from a disruption of the gonadotropins-releasing hormone (GnRH) secretion pattern. In healthy women, the ratio of LH to FSH typically ranges between 1 and 2. This ratio can sometimes be as high as 2 or 3 in women with polycystic ovarian condition.[6] The primary factor causing PCOS is an increase in the gonadotropin hormone, which causes an increase in LH and induces the production of more androgens (often testosterone) from the ovaries.[7] Most plants have been shown to have antiprotozoal (*T. gondii*) and against organisms such as Matricaria chamomilla.[8,9]

Materials and Methods

Study population

This study was conducted in Kirkuk city in two hospitals (Gynecological & Pediatric Hospital and Kirkuk General Hospital) on 200 aborted women aged (15-45 years) from November 1, 2022 to March 21, 2023.

Sample collection

From each aborted woman, 5 mL of venous blood was obtained using a sterile syringe, and we transferred 3 mL of that blood sample into sterile gel tubes. To allow the blood to coagulate, test tubes were left at room temperature for 20-30 min. The blood was separated by centrifuging it for 5–10 min at 3000 rpm. Following that, the serum was separated into four Eppendorf tubes and stored in a deep freezer (-20ºC) until needed. The serum was then used for the estimation of LH and FSH levels using a Cobas immunoassay analyzer.

Methodology

Cobas e411 immunoassay analyzers

Using Elecsys and Cobas e411 immunoassay analyzers, the electrochemiluminescence binding assay was designed to estimate LH and FSH which was done by using LH cobas kit, Roche, Germany and FSH cobas kit, Rorche, Germany according to the protocols designed by the manufacture of the diagnostic kits.

Ethical approval

The participants were given a description of the objective and process for gathering information from abortive women who visit hospitals at the beginning of the study after we received official approval ethical to conduct the study from Directory of Health/Kirkuk according to the document number 964 date October 25, 2022.

Statistical analysis

The data were analyzed according to Minitab statistical program with ANOVA test and *t* test. Duncan's multiple range test was used to compare the differences between means under the probability level 0.05 ($P < 0.05$).

Results

Out of 200 aborted women, 40 (20%) of them were PCO positive (poly cystic ovarian syndrome) and 160 (80%) were negative. The 40 positive PCO were divided into four groups: PCO +ve and Toxo IgM +ve (7.5%), PCO +ve and Toxo IgG +ve (15%), PCO +ve and Toxo IgM -ve (42.5%), PCO +ve and Toxo IgG -ve (35%) [Table 1].

Table 2 demonstrates the level of LH in four groups the highest mean was in Toxo IgM and PCO +ve (9.190 ± 3.710) in this table there were significant differences between the groups with *P* value of 0.046.

Table 3 demonstrates the level of FSH in four groups the highest mean was in Toxo IgG +ve and PCO +ve (8.776 ± 2.061) and the lowest mean was in Toxo IgM +ve

Table 1: The number and percentage of the four groups included in our study

Study groups	No.	%
Toxo IgM +ve and PCO +ve	3	7.5%
Toxo IgG +ve and PCO +ve	6	15%
Toxo IgM –ve and PCO +ve	17	42.5%
Toxo IgG –ve and PCO +ve	14	35%
Total	40	100%

Toxo IgM: toxoplasma immunoglobulin M, Toxo IgG: toxoplasma immunoglobulin G, PCO: polycystic ovarian syndrome

Table 2: LH level

Study groups	No.	LH level mIU/mL (mean ± SD)	*P* value
Toxo IgM +ve and PCO +ve	3	9.190 ± 3.710[a]	0.046
Toxo IgG +ve and PCO +ve	6	7.860 ± 3.110[b]	
Toxo IgM –ve and PCO +ve	17	6.707 ± 3.255[b]	
Toxo IgG –ve and PCO +ve	14	6.746 ± 3.500[b]	

[a, b] Different small letters refers to the presence of significant differences between groups at the level ($P < 0.05$)

Toxo IgM: toxoplasma immunoglobulin M, Toxo IgG: toxoplasma immunoglobulin G, SD: standard deviation, LH: luteinizing hormone, PCO: polycystic ovarian syndrome

Table 3: FSH level

Study groups	No.	FSH level MIU/mL (mean ± SD)	*P* value
Toxo IgM +ve and PCO +ve	3	5.790 ± 3.25[b]	0.045
Toxo IgG +ve and PCO +ve	6	8.776 ± 2.061[a]	
Toxo IgM –ve and PCO +ve	17	7.779 ± 3.017[a]	
Toxo IgG –ve and PCO +ve	14	6.927 ± 3.295[ab]	

Toxo IgM: toxoplasma immunoglobulin M, Toxo IgG: toxoplasma immunoglobulin G, SD: standard deviation, FSH: follicle stimulating hormone

[a, b] Different small letters refer to the presence of significant differences between groups at the level ($P < 0.05$)

and PCO +ve (5.790 ± 3.25) in this table there were significant differences between the groups with *P* value of 0.045.

Discussion

Toxoplasma gondii can cause congenital disease if it infects pregnant woman, and women are more susceptible than males to contracting *T. gondi.* The susceptibility to pathogens differs depending on the stage of the menstrual cycle in non-pregnant women as well as the stage of gestation. Public health officials are particularly interested in how sex and pregnancy-related hormones affect the severity of *T. gondii* infection.[10] According to a recent study Toxoplasmosis can affect FSH and LH and cause changes in hormone secretion during pregnancy. In general decline in gonadotropin hormone levels (FSH and LH) is associated with immune system issues and increased susceptibility to parasitic diseases.[11]

The pituitary gland releases the glycoproteins LH and FSH, which are then carried by the bloodstream to their sites of action (testes). GnRH promotes the release of both the FSH and LH hormones. Leydig cells secrete more testosterone as a result of LH hormone stimulation. Premature menopause, inadequate ovarian reserve, and testicular failure are just a few of the illnesses that can develop when the level of FSH is high, while obesity, polycystic ovarian syndrome, infertility, and gonadotropin deficit are caused by low levels of FSH.[12]

In our study we found significant differences at ($P > 0.046$) between groups, with a drop of LH in aborted women with acute infection (IgM Ab) and aborted women with chronic infection (IgG Ab). This result was agreed with.[13]

In addition in the current investigation, there were significant differences in the mean of FSH concentration with *P* value ($P < 0.05$) in group (Toxo IgG +ve and PCO +ve) (8.776 ± 2.061) compared with the lowest mean of (FSH) in Toxo IgM +ve and PCO +ve (5.790 ± 3.25).

According to Terpsidis *et al.*[14] and Dong *et al.*[15], the first theory suggests that *T. gondii* may stimulate the hypothalamic-pituitary-adrenal stress axis, which would then modify the hypothalamic-pituitary-gonadal axis, resulting in altered gonadotropin release.

Because some sex hormones are associated with immune response, sex hormones in high concentration are associated with low immune response, which may increase susceptibility to parasitic infection, the reason for the hormone's high level in infected women.[13]

Conclusions

PCOS, which is associated with infertility, is the most prevalent hormonal issue in women of reproductive age. In this study, we found that PCOS had a comparatively high frequency compared to the general population; its prevalence seems to be slightly higher in women who frequently miscarry. The levels of LH and FSH in our research groups were significantly different.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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