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Survey of toxoplasmosis, blood type, hematology,
pregnancy and abortion in outpatient women.

Noor H Hanoush Al-Fahdawi and Thaer A Salih Al-Aloosi.

Dept. Biology, College of Science, University of Anbar, Iraq.

ABSTRACT

Hanoush Al-Fahdawi NH, Salih Al-Aloosi TA., Survey of toxoplasmosis, blood type, hematology, pregnancy and abortion in outpatient women Onl J Vet Res., 23 (7):666-674, 2019. Authors surveyed toxoplasmosis, blood types and hematology in 120, 15-48 year old women outpatients from various hospitals in Iraq from October 2016 to January 2017. Blood samples were typed and *Toxoplasma gondii* diagnosed by ELISA. We found overall incidence of 56.6% and no relation with age or locality. Those with 3 abortions were 91% positive to *T Gondii*, first trimester with abortion 60%. blood group B ~46% and group O ~31%. We found differences ($P < 0.03$) in differential count of leukocytes, PCV and MCHc but none with Hb, PLT, RBC and MCH between patients and controls.

Key Words: toxoplasmosis, blood types, hematology, pregnancy, abortion, women.

INTRODUCTION

Toxoplasma gondii an obligate intracellular parasite induces a wide range of clinical symptoms related to immune status of patient (Norouzi et al., 2017; Olaniyan et al., 2019). (Smith, 2007). Toxoplasmosis causes abortion, congenital defects and blindness in acute or chronic forms (William and Demure, 2000). *T. gondii* can be carried by birds, mammals, and humans) (Dubey et al, 2007). In humans acute infection occurs by ingestion of parasite eggs (Al-Wattari, 2005) and tachyzoites in epithelial cells spread through blood and lymph and reach other tissues where they divide every 5-12 hours (Othman, 2004). Sixteen to 32 parasites can destroy host cells and in the chronic stage form cysts in brain,

retina, heart muscle and other muscles and are not readily visualized (Olaniyan et al., 2019). Robert-Gangneux and Dardé, (2012) found that chronic infection induces cellular immunity sufficient to break the cyst. Congenital toxoplasmosis may involve eyes, brain, kidneys, heart, liver, blood or spleen of offspring causing long term blindness, seizures, cerebral palsy, mental retardation and deafness (Jones, 2001).

Filisetti and Candolfi, (2004) reported that natural killer (NK) cells, macrophage and cytokines induce innate immunity against *T. gondii*. In the acute stages macrophages phagositize *T. gondii* (Wilson *et al.*, 2008). We surveyed 120 outpatient women suspect with toxoplasmosis for presence of *T. gondii* and possible correlations with blood type, hematology, pregnancy and abortion. Blood samples were taken from 60 normal women for comparison.

MATERIALS AND METHODS

Blood was taken from 120 women with a history of abortion and suspected toxoplasmosis and 60 healthy women aged 15-48 years. The samples were taken October 2016 to January 2017 at Obstetrics and Gynecology, Al-Nuaman hospital, Baghdad and The Teaching Al-Ramadi Hospital for Children and Gynecology, Iraq. Before collections, name, age, residency, occupation and number of abortions were recorded. Radial vein 2ml blood was taken in EDTA tubes for hematology (Henry and Davidson, 1974, Sood 1985) and 3ml serum for ELISA for toxoplasmosis (Engvall and Perlmann, 1971; Remington and Klein, 1976). Blood groups (Anti A, B and Anti-D slide tests were done as described by Issitt, (1985) and Anti- A, B, A+B and anti-D with a kit (Spinreact, Spain). Statistical differences between means were determined by ANOVA and Duncan's multiple tests ($P < 0.05$) (Leech et al., 2011).

RESULTS

Results are shown below in Tables 1-9

Table 1. *T. gondii* and abortion.

Abortions	Patients		
	Tested	<i>T. gondii</i>	%
None	32	13	40.6 ^{cd}
1	30	15	50 ^c
2	27	14	51.8 ^c
3	12	11	91.6 ^a
≥4	19	15	78.9 ^{ab}
Total	120	68	37.7
$P < 0.004$			

*different letters (a,b..ect) significant difference at $P \leq 0.05$

Table 2. *T gondii* and trimester of pregnancy.

Pregnancy		Patients		
		Tested	<i>T Gondii</i>	%
Trimester of pregnancy	3 months	60	36	60 ^a
	6 months	36	19	52.7 ^b
	9 months	24	13	54.1 ^b
	Total	120	68	37.7
P < 0.002				

*different letters significant difference (P ≤ 0.05).

Table 3. Toxoplasmosis and blood group types.

		Patients						
		Tested	<i>T Goondii</i>	%		Tested	<i>T gondii</i>	%
Blood group	A	45	18	40 ^{ab}	A+	26	11	42.3 ^b
					A-	19	7	36.8 ^c
	B	39	18	46.1 ^a	B+	21	8	38 ^{bc}
					B-	18	10	55.5 ^a
	AB	34	13	38.2 ^b	AB+	20	6	30 ^{cd}
					AB-	14	7	50 ^{ab}
	O	62	19	30.6 ^{bc}	O+	43	12	27.9 ^d
					O-	19	7	36.8 ^c
Total		180	68	37.7		180	68	37.7
P value 0.035								

*Different letters significant difference (P ≤ 0.05).

Table 4. Mean ± SD hematology in women with or without *T gondii*.

Group	N	RBC 10 ⁶ /μL	PCV %	MCV fL/RBC	PLT 10 ³ /μL	Hb g/dL	MCH pg/RBC	MCHc g/dL
Pregnant controls	30	4.30±.39 ^a	34.91±4.74 ^b	81.59±7.37 ^a	260.14±75.32 ^a	11.85 ± 2.04 ^a	27.74±3.75 ^a	33.78±2.04 ^{ab}
Healthy controls	30	4.64±.46 ^a	39.11±2.82 ^a	84.75±6.41 ^a	200.94±88.73 ^a	12.94 ± 1.32 ^a	28.27±2.94 ^a	33.24±1.62 ^{ab}
Infected with IgG	29	4.47±.97 ^a	39.06±5.34 ^a	90.3±7.6 ^a	249.80±56.32 ^a	12.81 ± 2.33 ^a	28.25±1.75 ^a	32.10±3.25 ^b
Uninfected abortive	42	4.48±.43 ^a	37.45±3.93 ^{ab}	85.14±7.91 ^a	226.58±77.76 ^a	12.33 ± 1.70 ^a	28.02±3.61 ^a	32.85±1.99 ^{ab}
Infected with IgM & IgG	23	4.31±.41 ^a	37.47±4.89 ^{ab}	87.64±5.64 ^a	248.91±45.96 ^a	12.96 ± 1.79 ^a	30.39±2.16 ^a	34.35±1.52 ^a
Infected with IgM	26	4.59±.36 ^a	39.38±3.90 ^a	85.13±5.89 ^a	254.00±81.28 ^a	12.57 ± 1.28 ^a	28.40±3.27 ^a	32.71±1.64 ^{ab}
Total	180	4.47±.50	37.76±4.28	180.47±94.1	234.34±76.15	12.52 ± 1.73	28.35±3.21	33.13±2.06

*Different letters significant difference (P ≤ 0.05).

Table 5. Mean ± SD total and differential WBC (%) in women with or without *T goondi*.

Group	N	White cells *10 ³ /μL	Neutrophils	Eosinophils	Basophils	Lymphocytes	Monocytes
Pregnant controls	30	9.78±2.44 ^{ac}	68.23±5.83 ^b	1.71±0.97 ^a	1.05±1.84 ^a	23.44± 5.34 ^b	5.14±1.25 ^{ac}
Healthy controls	30	6.80±1.57 ^b	47.07±17.17 ^a	2.60±3.67 ^a	2.09± 77.42 ^a	32.46± 12.91 ^a	5.97±2.57 ^{ab}
Infected with IgG	29	10.19±3.32 ^a	70.53±9.74 ^b	1.76± 1.46 ^a	.87± .71 ^a	20.84± 9.11 ^b	4.25±1.61 ^c
Uninfected abortive	42	9.10±2.80 ^{ac}	67.81±11.23 ^b	2.06±1.72 ^a	.77± .42 ^a	23.57±9.77 ^b	5.77±1.65 ^{abc}
Infected with IgM & IgG	23	10.27±2.77 ^a	70.62±6.05 ^b	2.03±1.36 ^a	.56±.25 ^a	21.77±4.91 ^b	5.03±1.32 ^{ac}
Infected with IgM	26	7.96±2.46 ^{bc}	64.24±10.99 ^b	1.60±1.35 ^a	.83± .32 ^a	27.18±9.40 ^{ab}	7.02±2.27 ^b
Total	180	8.90±2.79	64.29±13.95	2.03±2.05	5.82± 34.12	25.07±9.99	5.60±1.93

*Different letters significant difference (P ≤ 0.05).

Table 6. Incidence of *Toxoplasma* in women by age.

Patients			
Age.	Tested	Positive	%
< 20	30	12	40.0 ^{ab}
20-29	62	27	43.5 ^a
30-39	47	17	36.1 ^b
≥ 40	41	12	29.2 ^c
Total	180	68	37.7
P value 0.008			

*Different letters significant difference ($P \leq 0.05$).

DISCUSSION

Table 1 lists toxoplasmosis and number of abortions showing that women with 3 or more abortions 91.6% tested positive and with 1 to 2 ~50% and none 40.6% ($P < 0.05$). Al-Hussien (2016) found that women with high IgM levels could suffer 3 or more miscarriages (10.71%) compared with other groups. Kreem, (2012) reported high incidence (80.6%) in women with single or repeated abortions. In Jordan, Abu-Madi et al., (2010) found that women with 3 to 7 miscarriages were twice as likely to test seropositive for *T. gondii* compared with women with normal pregnancies. In contrast, Saif *et al.*, (2014) reports that women with 1 abortion had higher anti-toxoplasma antibodies when not pregnant (46.15 %) or pregnant (50%). Othman, (2004) also found that higher seropositive in pregnant women with history of one abortion (55.17%) compared with 2-4 (50%) or 5-8 (33.33%). We surmise that higher incidence occurred in women with 3 abortions as they may be infected before the first pregnancy, as well as 2nd and 3rd time after abortion and do not get tested for treatment (Karem, 2007; Kreem, 2012).

We found similar incidence (~53-60%) for toxoplasma during all trimesters (Table 2). Our results contrast with Al-Ubbyde, (2004) who reported 76% or Al-Azawi, (2015) 73.6% during the 1st trimester. During the 3rd trimester, Al-Husseini et al., (2016) reported ~88% but Sefah-Boakye, (2015) only 9.73% and Al-Hussien et al, (2016) only 11.4% and 1.3% in 2nd and 3rd trimesters. Norouzi *et al.*, (2017) reported significant association between seropositivity and 2nd trimester, a finding replicated by Al-khafaji *et al.*, (2011). Al-Azawi, (2015) maintains that toxoplasmosis is more severe during the 1st months of pregnancy since 10-20% fetuses may be infected inducing spontaneous abortions. According to Al-Hussien et al., (2016), in most cases, women abort at end of 1st trimester or beginning of the 2nd

Results on Table 3 suggest highest prevalence of *T gondii* in women with blood group B (46.1%) and lowest with O type (30.6%) ($p < 0.05$) with B- 55.5% and O+ and AB+ only 27.9% and 30%. AL-Taei, (2015) found 37% in patients with B type and lowest in type AB. AL-Ghezy, (2012) found 32-35% type B or O incidence in women with toxoplasma that had aborted. Al-Shikhly (2010) reported 30.6% in females with B type. Abdullhusein, (2017) found that AB blood types has high seropositivity to anti-*Toxoplasma* Abs. Kolbekova,

(2007) suggested a relationship between ABO groups and anti-*T. gondii* antibodies. AL-Taei, (2015) maintained that antigen B on RBC surface may be a receptor for *T. gondii*.

Our findings showed no differences in RBC, PCV, MCV, Hb, MCH or platelets between any cohort (Tables 4) However we did find higher ($P \leq 0.05$) MCHC in infected women with IgM & IgG (34.35 ± 1.52) but lower in infected women with IgG (32.10 ± 3.25) compared with other groups. Generally Hb was higher in controls (12.94 ± 1.32 g/L) but lower in healthy pregnant women (11.85 ± 2.04 g/L). Ali, (2011) reported no differences in RBC but found reduced PCV in pregnant or aborting women. Al-Jabiry, (2010) reported reduced RBCs, Hb, PCV during 3rd trimester compared with non-pregnant women. Al- Abaas *et al.*, (2015) found decreased Hb and RBCs in patients with *T gondii*. Al - Delamy, found no effect on platelets. Hematology might not change in pregnant infected women with toxoplasmosis as (Husain and Abdulatef, 2014). Possibly reduced Hb in infected women may be due to iron loss (Javadj *et al.*, 2010).

As expected, we found increased WBC in infected women (Table 5) with $\sim 10 \times 10^3/\mu\text{L}$ in IgG and IgG & IgM compared with controls $\sim 7 \times 10^3/\mu\text{L}$ or women infected with IgM $7.96 \pm 2.46 \times 10^3/\mu\text{L}$. Lymphocytes were higher ($P 0.05$) in controls (32.46 ± 12.91) and infected women with IgM was ($27.18 \pm 9.40\%$) whereas monocytes were increased in infected women IgM ($7.02 \pm 2.27\%$) but lower in infected women with IgG ($4.25 \pm 1.61\%$) compared with others groups. Neutrophils were higher in controls (47.07 ± 17.17) compared with other groups of women but we found no differences with basophil and eosinophils.

Al-Abaas *et al.*, (2015) and Al-Ghezy, (2012) reported similar findings with WBCs in patients with *Toxoplasma gondii* compared with controls. Ajeel, (2012), however reported reduced WBC but in infected rats. In the main, most authors found little or no differences in WBC, lymphocyte, basophils, eosinophils, monocyte, neutrophils between *T gondii* patients and controls (Al-Delamy, 2002; Al-Mosawi, 2014). We expected an increase in WBC, IgM and IgG to be induced by infection by *T. gondii*, as reported by others (Al- Abaas *et al.*, 2015) and (Al-Ghezy, 2012). In chronic infections, tachyzoites do not replicate and cysts may not elicit immunity, symptoms or changes in WBC (Al-Jabiry, 2010).

We found most infection in 20-29 year olds accounting for 43.5% and least in those 40 or older (Table 6). Our results replicate those of Al-Ghezy, (2012); Al- Dahmoshi *et al.*, (2013); Sefah-Boakye, (2015); Imam *et al.*, (2016); Al-Hussien *et al.*, (2016) and (Norouzi *et al.*, (2017) in Iraq. Mohammadi *et al.*, (2015) reported highest incidence in 28-33 year olds, but no significant differences amongst them whereas Kreem, (2012) at 40-49 years. Prevalence of *T. gondii* may increase with age (Matowicka-Karna *et al.*, 2009). In our study, the high prevalence in young adult women, may be due to frequent exposure to infective stages of the parasite (Imam *et al.*, 2016). We found no differences in *T gondii* infection between rural or urban women (both $\sim 38\%$). Similar findings have been published (Othman, 2004); (A'aiz, 2010); Al-(Saadii, 2013); (Darweesh *et al.*, 2018).

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