

Study the Relationship of Toxoplasma IgG Titer with Some Other Immunoglobulin and Complement Components

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Abstract:

Background: Toxoplasmosis is caused by intracellular protozoan parasite; it is difficult to distinguish between the recently acquired infection and the chronic infection because of the frequent presence of *Toxoplasma* antibodies in the general population.

Objective: This study was conducted to estimate the relationship between the *Toxoplasma* antibodies titers and other immunoglobulin and complement components

Study design: Sera from 40 Iraqi blood donors of different ages (18-45 years) during first of February- first of December /2008 in Baghdad city, were screened for *Toxoplasma* antibodies titers (28 positive and 12 negative). Agglutination method was used according to the kit procedure (Toxo Cell Latex) and single radial immunodiffusion plates were used for estimation of IgM, IgA, C3 & C4.

Results: The results showed a significant differences ($P<0.01$) in the all means of IgM in all titers compared to controls, and the highest titer of IgM (197 ± 21.27 mg/dl) has been shown at a titer of IgG 160 IU/ml. There was no significant differences ($P>0.05$) shown between IgA titers compared with IgG titers, while there were a high significant differences ($P<0.01$) of C3 & C4 compared with the IgG titers at level 10, 20&10 IU/ml respectively.

Conclusion: The results indicated that there is a relationship between titers of IgG and titers of IgM and complement components (C3 & C4) during the course of *Toxoplasma* infection especially at the beginning and chronic infection when the level at IgG titers was low.

Key words: Toxoplasmosis, IgG, IgM, C3, C4

Introduction:

Toxoplasmosis is a worldwide Zoonosis caused by a protozoan parasite. *Toxoplasma gondii* infection is usually benign, acquired through ingestion of oocysts excreted in cat feces or through ingestion of tissue cysts in under cooked meats, more serious forms of the disease is congenital Toxoplasmosis transmitted from mother to fetus through the transplacental route^[1]. The disease may be transmitted by blood transfusion^[2].

The clinical manifestations of Toxoplasmosis result from direct tissue destruction by the parasites, but inflammatory cytokine mediated immunopathological changes may also contribute to disease progression^[3]. And immune suppression by retroviruses, pregnancy and corticosteroid therapy are amongst the fetus influencing the clinical course, immune response, treatment response and prognosis of clinical Toxoplasmosis in cat and man^[4].

In general, antibodies serve to control the level of parasites that exist free in the blood stream and tissue fluids, where as cell mediated immune responses are directed largely against intracellular parasites, antibodies together with the complement and cytotoxic cells may kill them. Both antibody-mediated and cell-mediated immune responses occur following infection with

Toxoplasma; the antibodies acting in conjunction with complement can destroy organisms found free in body fluids but they naturally have little or no influence on the intracellular forms of the parasites, for that *T. gondii* tachyzoites can transform themselves into a cyst form containing bradyzoites, the cyst appear to be non immunogenic and do not stimulate an inflammatory response (not recognized as a foreign)^[5].

This study was conducted to estimate the relationship between the *Toxoplasma* antibodies titers and other immunoglobulin and complement components.

Materials & Methods:

1- Blood Samples:

Sera from 40 Iraqi blood donors of different ages (18-45 years) during first of February-first of December 2008 in Baghdad city were screened for *Toxoplasma* antibodies titers (28 positive & 12 negative). Agglutination methods were used for determination of antibody titers; by using the dilution with normal saline (NaCl 9%). As follow: 1:1, 1:2, 1:4, 1:8, 1:16, 1:32; which equal to 1:10, 1:20, 1:40, 1:80, 1:160, 1:320 respectively according to kit procedure*

2-Immunoglobulines and Complement components

Single radial immunodiffusion plate was used for determination of IgM, IgA, C3 & C4 according to kits procedure**

3- Statistical Analysis:

Statistical analysis was conducted to describe different variables and parameters in current study and to describe relationships with each other as well. Independent t-test of significance was used for two-group comparison [6].

Results:

1- IgM:

The results show significant differences ($P < 0.01$) in the means of IgM in all titers compared to controls of different titers of IgG (1:10, 1:20, 1:40, 1:80, 1:160, 1:320) and the highest titer of IgM (197 ± 21.27 mg/dl) has been shown at a titer of IgG 160 IU/ml (Table.1).

2- IgA:

There were no significant differences ($P > 0.05$) shown of IgA titers compared with control group and the highest titer (310.65 ± 29.65 mg/dl) at a level of IgG titer 10 IU/ml (Table. 1).

3- Complement components

There was a highly significant differences ($P < 0.01$) shown at a levels of IgG 10&20 IU/ml of C3 (323.00 ± 23.1 mg/dl and 96.30 ± 14.70 mg/dl) respectively in comparison to the control group which showed a titer 179.76 ± 11.85 mg/dl. While there were no significant differences ($P > 0.05$) of the remaining titers of IgG titers 40,80, 160, 320 IU/ml.

The C4 showed a significant differences ($P < 0.01$) at a level of 10 IU/ml of IgG which show a titer 23.70 ± 6.6 mg/dl. compared to control group which showed a level of 53.94 ± 6.88 mg/dl (Table.1).

Table 1: The relationship between the titers of IgG and IgM, IgA and complement components (C3& C4).

No. of cases	IgG IU/ml	IgM mg/dl	IgA mg/dl	C3 mg/dl	C4 mg/dl
2	10	* 167.25 ± 9.9500	310.65 ± 29.65	* 323.00 ± 23.10	* 23.70 ± 6.60
2	20	* 194.50 ± 3.50	253.55 ± 27.45	* 96.30 ± 14.70	46.15 ± 8.35
3	40	* 184.50 ± 14.46	236.33 ± 84.88	208.93 ± 54.64	46.63 ± 9.67
8	80	* 179.40 ± 8.40	307.02 ± 23.93	212.23 ± 32.80	54.61 ± 5.38
7	160	* 197.51 ± 21.27	352.84 ± 30.54	190.11 ± 31.28	55.64 ± 7.02
6	320	* 162.83 ± 13.88	271.20 ± 46.57	199.60 ± 31.97	56.95 ± 9.23
12	Control	88.43 ± 6.44	316.49 ± 48.98	179.76 ± 11.85	53.94 ± 6.88

* $P < 0.0$

*Toxocell latex **Biomaghreb IgM844-IgA868- C₃ 785- C₄ 768

Discussion:

It is difficult to distinguish between recently acquired infection & chronic infection because of the frequent presence of *Toxoplasma* antibodies. These antibodies may persist in the serum of an a symptomatic individual for years at high titers [7]. These findings conducted our results. The transient rise of IgM represented immune responses to the highly invasive blood or lymph-borne tachyzoites. The immune responses were strong enough that the tachyzoites replication is attenuated to become the slow replicating and possibly encysted bradyzoites which are located in the muscle, viscera or central nervous system with lower antigenicity [4]. The tendency of specific IgM persists for long time, many years after the acute infection, even at high levels [8]. In addition

the anti-toxoplasma antibody remains detectable throughout the life time of the host. [9]. Also the presence of the parasite in a parasitophorous vacuole enables it to escape the humoral immune defenses [10].

Complement components are synthesized at various sites throughout the body; C3&C4 are synthesized also by macrophages. These proteins are readily available for defense at sites of inflammation where macrophages accumulate; IgG is much less active than IgM in activating complement. C3 is the highest concentration in the serum which is activated commonly by classical and alternative pathway; the activation of C3b by C4b2b is a major step in the complement activation process because each C4b2b complex can activate as many as 200 C3 molecules [5].

These attributed reflexes on our results that showed significant values in IgG titers 10&20 IU/ml on the levels of C3&C4 which could explain a flaring up of the parasite or acute phase of infection.

Our conclusion shows there is harmony synchronization between the IgG, IgM&C3, C4 titers, while there is no effect on the IgA titers during the course of *Toxoplasma* infection.

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