

Role of *Toxoplasma gondii* as a risk factor for heart disease and its relationship to some physiological criteria

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Abstract

The study aimed to find out the effect of infection with *Toxoplasma gondii* as a risk factor for heart disease (tachycardia) with the effect of infection on lipid levels in the blood serum, in the period from October 2021 until February 2022. 200 blood samples were taken from patients who visited the outpatient clinics of cardiologists and Samarra General Hospital. The samples were divided into three groups, the first were those with heart disease and *Toxoplasma*, the second were those with heart disease only, and the third was the control group. The separated serum was kept at a temperature of (-20) until use. Enzyme-linked immunosorbent assay (ELISA) was used to detect toxoplasmosis. Lipid profile levels were measured using the Spectrophotometric method.

The results of the study showed a significant increase at a significant level ($P < 0.05$) in the levels of cholesterol, triglycerides and HDL. In the group infected with *Toxoplasma* compared to the control group. On the other hand, it was found that there were no significant differences in cholesterol and triglyceride levels between the group infected with *Toxoplasma* and the group with heart disease only, and there were no significant differences in LDL levels in the three study levels.

Keywords: *Toxoplasma gondii*, cardiac diseases, physiological criteria.

INTRODUCTION

Toxoplasma gondii is a parasitic protozoan that can infect many host species of mammals and birds, including humans, causing toxoplasmosis. Members of the Felidae family, which includes cats, are important final hosts of the sexual phase (La Hoz et al. 2019). It can infect many intermediate hosts such as rodents, birds, farm animals and wild animals as well as humans (Jungersen et al., 2001; Wilson and McAuley, 2009). *Toxoplasma gondii* is one of the protozoa that is obligatory intracellularly parasitic (Dubey, 2010; Jimenez-Coello et al, 2012).

Cardiovascular disease is the most common cause of death and is responsible for about 30% of deaths worldwide (Sans et al., 1997). Palpitations are one of the most common complaints of patients who present to emergency departments, primary care providers or cardiologists. One study indicated that 16% of patients who presented to the primary care center had heart palpitations (Weitz and Weinstock, 1995).

Studies have shown that patients with chronic cardiovascular disease are often exposed to opportunistic infections such as toxoplasmosis due to the general body weakness and immunodeficiency state. In a 2006 study conducted in Mexico, a significant relationship was found between toxoplasmosis and chronic cardiovascular disease (Alvarado-Esquivel et al, 2016). Undoubtedly, the socioeconomic toll caused by this disease along with the transformations caused by this parasite In the human body it can be costly to society compared to other diseases (Yousefi et al, 2017).

The study aimed to know the role of *Toxoplasma condensate* as a risk factor for heart disease and its relationship to some physiological criteria.

Materials and methods:

1- Sample collection: the study included the collection of 200 serum samples collected during the period from (October 2021) to (February 2022). Airtight without anticoagulants, centrifuge for 10 minutes at 3000 rpm, after leaving tubes at room temperature 25°C until coagulation. Then the serum was withdrawn by micropipette and placed in clean, sterile tubes and kept in a frozen state at (-20) °C until the tests under study were carried out.

2- Study design: The study groups were divided into three groups: (with heart disease and Toxoplasma, patients with heart disease only, control group).

To verify the class of IgM and IgG antibodies to Toxoplasma, ELISA was used using the standard method manufactured by (German Human). Lipid profile levels were measured by the Spectrophotometric method.

3- Statistical analysis: The results obtained from the current study were analyzed using the SPSS statistical program, and Dunkin' polynomial test was used to compare three groups at the level of differences between the averages of the totals.

Results and discussion: The results show an increase in the levels of CHI, TG and HDL, 223.10 ± 11.77 , 208.80 ± 17.72 , 39.459 ± 5.298 , respectively in the group infected with toxoplasmosis and heart disease compared to the control group 207.69 ± 18.08 , 170.28 ± 18.88 , 35.640 ± 6.52 , respectively, with no significant difference at ($P \leq 0.05$) in LDL levels 141.93 ± 20.67 compared to the control group 138.76 ± 14.98 . In the group of patients with heart disease only, there was an increase in the levels of CHI, TG and HDL. 222.08 ± 19.10 , 205.57 ± 18.62 , 37.744 ± 6.368 , respectively, compared to the control group 207.69 ± 18.08 , 170.28 ± 18.88 , 35.640 ± 6.52 , respectively, with no significant difference at ($P \leq 0.05$) in LDL levels 142.94 ± 17.03 In this group compared to the control group 138.76 ± 14.98 Table (1).

Table (1): lipid profile level in study groups.

Groups Parameters	Control Mean± St.d	Toxoplasma &CVD Mean± St.d	CVD Mean± St.d	P-Value
CHI (ng/ml)	207.69±18.08 b	223.10±11.77 a	222.08±19.10 a	0.05 *
TG (ng/ml)	170.28±18.88 b	208.80±17.72 a	205.57±18.62 a	0.0006 **
HDL (ng/ml)	35.640±6.52 b	39.459±5.298 a	37.744±6.368 ab	0.012 **
LDL (ng/ml)	138.76±14.98 a	141.93±20.67 a	142.94±17.03 a	0.874

This study showed remarkable results confirming that *T. gondii* affects the tachycardia index in infected persons. This confirmation was confirmed by the lipid profile picture, which revealed an increase in cholesterol and triglycerides and a triglyceride status characterized by a significant increase in cholesterol levels and a small percentage increase in HDL. This relationship between blood cholesterol levels in a human infected with parasites has drawn the attention of various workers. Toxoplasma cannot synthesize new cholesterol and depends on the acquisition of LDL-derived cholesterol from the host cell, by cellular endocytosis mediated by the LDL receptor, or LDL receptor-binding protein, Entry of Toxoplasma into cells (Coppens and Joiner,2003).

These studies indicated that cholesterol has an important role in the pathogenesis of toxoplasmosis. However, data regarding the sources of parasite lipids are scarce and the molecular mechanisms by which Toxoplasma acquires lipids from host cells is largely unknown (Coppens et al, 2006). Or, this rise in the level of CH could be attributed to several reasons, including the

decomposition of low-density lipoprotein LDL, or as a result of the inefficiency of the receptors for the protein part of LDL in tissues, as well as the activity of the enzyme Cholesterol acyl transferase, which is responsible for cholesterol absorption in the intestine, as well as an increase in its internal synthesis in addition. To consume meals rich in saturated fats. (Robak et al, 2004). With regard to the group infected with Toxoplasma and heart diseases with the group with heart diseases only, it was found through the current study that the toxoplasma parasite does not contribute to an increase in cholesterol, because there are no significant differences between the group infected with the parasite and heart disease than the group infected with heart disease only without a parasite infection, The results of the current study showed that there were significant differences in the levels of triglycerides between the control group, the group of people infected with Toxoplasma and heart diseases, and the group of people with heart diseases only.

The blood vessel, which makes it less in diameter, and thus reduces the amount of blood flowing to and from the heart, which leads to the occurrence of cardiovascular diseases, including tachycardia (palpitations), or it is possible that the increase in the level of TG in the blood serum is due to the increase in the activity of the enzyme lipase in Fat cells, causing an increase in the breakdown of stored fats and the release of large amounts of glycerol and acids Fatty acids into the blood, and when they were transferred to the liver, they were re-manufactured, and the increase in fatty acids in the liver transformed some of them into phosphorylated fats and glycerol, which were transferred with the triglycerides formed in the liver to the blood, thus resulting in an increase in the levels of fats in the blood (Nelson and Cox, 2005). Or, the high levels of TG in tachycardia patients may be attributed to metabolic problems in the body or to eating foods rich in fats (Solano and Goldberg, 2006). On the other hand, it was found that there were no significant differences between the group of people infected with Toxoplasma and heart diseases and the group of people with heart diseases only, which gives evidence that the Toxoplasma had no effect in causing an increase in triglycerides. With regard to HDL, the results of the current study agreed with the results of the study (Rimland et al, 2006), where there were significant differences between the three study groups.

The results showed that people infected with Toxoplasma parasite and heart diseases were significantly more than the group of people with heart diseases and the control group. So far, you know the mechanism of the effect of Toxoplasma in increasing the proportion of HDL. It is known that the high-density lipoprotein (HDL) and its function is to transport other fats in the blood plasma such as (LDL) (TG) from the blood vessels and deliver them to the liver so that the body can get rid of them and prevent their harmful effects, and this is what urges us There is a need for future studies to characterize the importance of the increase in HDL during infection with Toxoplasma for people with heart diseases. As for the percentage of LDL, it was found that there were no significant differences between the total of the three study. In conclusion, we should note that this study has shown that *T. gondii* had a role In changing the values of lipids in people with heart disease, which was characterized by an increase in cholesterol, HDL and TG, with no significant differences in the level of LDL.

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