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Biochemical, Physiological, and Immunological Changes Associated with *Toxoplasma gondii* Infection and Their Effects on Human Brain Health

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Abstract
Toxoplasma gondii is an intracellular protozoan parasite infecting a large percentage of the world's population and potentially staying a long time in human tissues, especially in the central nervous system. The long-term consequences of chronic infection have been linked to neuroinflammation, immune dysfunction, oxidative damage and neurological issues. This study proposed to examine the physiological and immunological changes resulting from infection with Toxoplasma gondii and to assess changes in brain health in humans.
In this cross-sectional study, the age range was 20-60 years and the subjects were divided into four groups: healthy control, acute toxoplasmosis, chronic toxoplasmosis and treated patients with anti-parasitic drugs. The parameters were assessed for physiological, hematological, immunological, oxidative stress and neuroimaging parameters. Neurological examination, cognitive assessment and magnetic resonance imaging (MRI) were used to assess brain involvement, and serum levels of pro-inflammatory cytokines, anti-Toxoplasma antibodies, and oxidative stress biomarkers were determined by standard laboratory techniques.
Particularly, the participants with infection showed substantial changes, as compared to healthy controls ($P < 0.05$). Increased levels of TNF-α, IL-6, IFN-γ, and anti-Toxoplasma antibodies were also observed in acute and chronic toxoplasmosis, along with increased levels of oxidative stress markers and decreased levels of antioxidant capacity. Hematological changes (increase in WBC and changes in immune cell populations) were also observed. MRIs showed different extents of brain involvement, especially in the patients with chronic toxoplasmosis. The levels of inflammatory cytokines correlated positively with neurological abnormalities.
The *Toxoplasma gondii* infection causes a number of physiological, immunological and neurological changes in humans. During chronic infection, the brain may be involved due to the persisting inflammation and oxidative stress. The knowledge of these mechanisms can help to better diagnose, monitor and treat toxoplasmosis and its neurological effects.

Keywords: *Toxoplasma gondii*; Human toxoplasmosis; Neuroinflammation; Immune response; Oxidative stress; Brain involvement; Magnetic resonance imaging (MRI); Neurological manifestations

1. Introduction

Toxoplasmosis is a common zoonosis that results from infection with an obligate intracellular protozoan parasite named *Toxoplasma gondii*, which can infect nearly all warm-blooded animals, including humans. It is regarded as one of the most successful human parasites worldwide as it has the capacity to establish acute and chronic infection and maintain itself in host tissues for prolonged periods. In immunocompetent people, infection is generally either asymptomatic or with mild symptoms, but can result in severe complications in immunocompromised people and pregnant women and in congenitally infected infants (Montoya and Liesenfeld, 2004; Elsheikha et al., 2020).

Humans can catch infection from eating oocysts in food or water that have been shed by infected cats or from eating undercooked meat that contains tissue cysts, or via congenital transmission from the mother to the fetus. Rapidly multiplying tachyzoites spread throughout the body and later transform into bradyzoites which form long-

term tissue cysts in various organs, including the brain, skeletal muscles and retina (Pan et al., 2022; Hakimi et al., 2021).

The infection of *Toxoplasma gondii* is a significant worldwide public health problem. Around one-third of the world's population is estimated to have been exposed to the parasite, though prevalence is very variable from region to region, depending on climate, dietary practices, socioeconomic status, hygiene and exposure to cats and contaminated soil (Wang et al., 2020). Infections are much more common in developing countries because sanitation is poor and people are exposed to contaminated environments (Rostami et al., 2020).

One of the key organs infected by chronic infection with *Toxoplasma gondii* is the CNS. Once the blood–brain barrier is crossed, the parasite may remain in the brain, leading to chronic neuroinflammation and changes in brain function. Persistent infection has been linked with cognitive impairment, memory loss, changes in neurotransmitter signaling and risk for multiple neuropsychiatric disorders (Zhao et al., 2022; Andreou et al., 2024; Mares et al., 2024).

A host immune response is very important in the control of parasite replication. Several pro-inflammatory cytokines such as interferon gamma (IFN-γ), tumour necrosis factor alpha (TNF-α) and interleukin-6 (IL-6) are secreted by innate and adaptive immune responses. These cytokines are crucial to control the proliferation of parasites, but chronic immune activation can lead to oxidative stress, damage to neurons and tissue, especially in the brain (Nasuhidehnavi et al., 2021).

Furthermore, the infection by *Toxoplasma gondii* could affect the physiological homeostasis through modulation of haematological parameters, antioxidant defence mechanisms and metabolic functions (Szewczyk-Golec et al., 2021). High levels of reactive oxygen species (ROS) may overwhelm the body's own antioxidant systems, causing lipid peroxidation, dysfunction of cells and neurological problems (Savadelis et al., 2023; Estato et al., 2023; Yang et al., 2024; Li et al., 2024; Paraboni et al., 2022).

Although extensive research has been conducted on toxoplasmosis, there is still a lack of research that has studied simultaneously the relationships among physiological changes, immune responses, oxidative stress biomarkers, and brain involvement in human patients. Previous studies were mostly conducted on the single manifestation of the disease and did not investigate the systemic and neurological function as a whole (Savadelis et al., 2023; Estato et al., 2023; Yang et al., 2024; Li et al., 2024; Paraboni et al., 2022; Yao et al., 2024).

The current study aimed to explore the physiology and immunology of *Toxoplasma gondii* infection in humans and the effects on the brain. Additionally, this study sought to establish the association between inflammatory response, oxidative stress markers, and neuroimaging aspects in patients with acute, chronic and treated toxoplasmosis (Zhao et al., 2022; Savadelis et al., 2023; Li et al., 2024).

2. Materials and Methods

2.1 Study Design

This cross-sectional clinical study aimed to explore the physiological and immunological alterations, which may occur due to *Toxoplasma gondii* infection, and how it might affect brain function in human beings over 6 months (Pleyer et al., 2020; Robert-Gangneux and Dardé, 2012).

2.2 Study Population

Sixty participants, aged 20 to 60 years were enrolled and assigned to four groups (n = 15 per group) (Rostami et al., 2020).
Group I: Healthy controls (n = 15)
Group II: Acute toxoplasmosis patients (IgM positive) (n = 15)
Group III: Chronic toxoplasmosis patients (IgG positive) (n = 15)
Group IV: Treated toxoplasmosis patients receiving anti-parasitic therapy (n = 15)

2.3 Inclusion and Exclusion Criteria

Inclusion criteria (Robert-Gangneux and Dardé, 2012)
Adults aged 20–60 years.
Laboratory-confirmed toxoplasmosis.
Capacity to give informed consent.
Exclusion criteria (Rostami et al., 2020)
Pregnancy.
HIV infection.
Autoimmune diseases.
Malignancy.
Immunosuppressive treatment.

2.4 Physiological Measurements

During standard clinical examination, physiological parameters such as body mass index (BMI), body temperature, blood pressure, and neurological symptom scores were measured according to standard procedures.

Body weight: The body weight was taken on a calibrated digital weighing scale with an accuracy of 0.1 kg with the participants wearing light clothes and without shoes.

Body temperature: During clinical evaluation, the body temperature was taken with a calibrated digital infrared thermometer or an oral digital thermometer.

2.5 Hematological Analysis

All participants had venous blood samples (5 ml) drawn with aseptics for hematological evaluation. The hematological parameters were analyzed by an automated hematology analyzer according to its manufacturer's instructions. The parameters chosen for evaluation of the systemic effects of *Toxoplasma gondii* infection on blood cell populations and the host immune response.

The following haematological indices were determined:

The levels of white blood cells (WBC): These are used to assess immune activation and inflammatory response as a result of infection.
The red blood cell count (RBC): was measured to determine the effect of the infection on erythropoiesis and oxygen transport.
Hemoglobin concentration (Hb): Test to ensure that there is not anemia or change in carrying capacity of oxygen in the system with systemic inflammation.
Platelet count (PLT): To evaluate changes in hemostasis and inflammation, if an infection occurs.
Neutrophil percentage: This represents a measure of the innate immune response, and is thought to represent the first line of defense against invading pathogens.
Breathing resistance cells: measure to evaluate adaptive immune responses; specifically to evaluate the activation of T lymphocytes regulating intracellular parasites.
Duplicate sampling was done for all laboratory analyses to ensure quality control. The values obtained were noted and expressed as (Mean ± SD) to carry out statistical analysis.

2.6 Immunological Assays (ELISA)

The samples in the serum were centrifuged at 3000 rpm for 15 minutes and then kept at −20°C for analysis. The concentrations of cytokines and antibody were determined by commercial ELISA kits in accordance with the manufacturer's instructions.
All of the above are:
Pro-inflammatory Cytokines: Tumor necrosis factor alpha (TNF-α), Interleukin-6 (IL-6) and Interferon gamma (IFN-γ).
Antibody that is anti-inflammatory: Interleukin-10 (IL-10)
Previously, anti-Toxoplasma antibodies, which included Immunoglobulin G (IgG) and Immunoglobulin M (IgM), were used.
The absorbance values were measured using an ELISA microplate reader at 450 nm.

2.7 Oxidative Stress Biomarkers

Previously described procedures were used to determine oxidative stress biomarkers. The following parameters were measured:
Malondialdehyde (MDA): Lipid peroxidation was assessed by measuring the MDA level.
The activity of the antioxidant defense enzyme superoxide dismutase (SOD) was measured.
Catalase (CAT): CAT activity was determined by the breakdown of hydrogen peroxide.

To assess the intracellular antioxidant status, reduced glutathione (GSH) level was determined.
The overall antioxidant defense system was evaluated as the total antioxidant capacity (TAC) (Szewczyk-Golec et al., 2021; Paraboni et al., 2022).

2.8 Brain Assessment

Neurological examinations, cognitive assessment tests and magnetic resonance imaging (MRI) were used to evaluate brain involvement when available. Brain abnormalities were assessed semi-quantitatively based on their degree of involvement, with 0 for no abnormalities (normal); 1 for mild abnormalities; 2 for moderate abnormalities; and 3 for severe abnormalities. Each participant's severity scores were collected to evaluate the relationship between neuroimaging, neurological symptoms, inflammatory cytokines and oxidative stress biomarkers.

2.9 Statistical Analysis

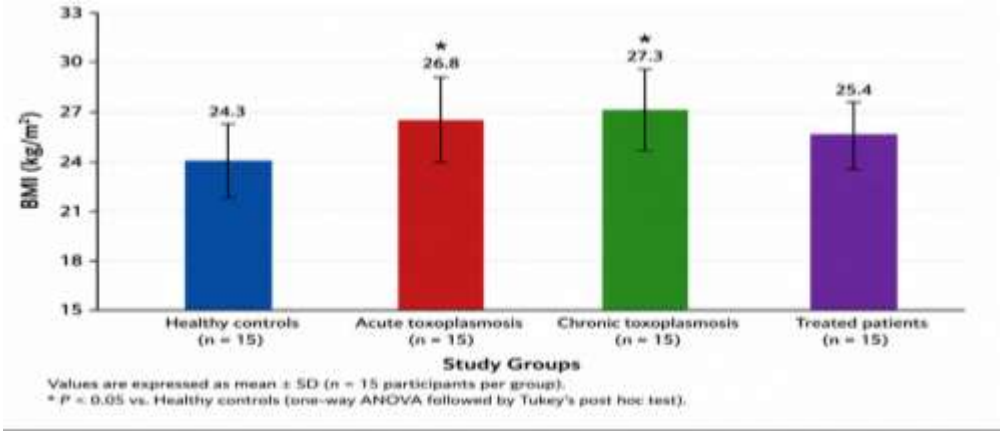
Data obtained were statistically analyzed using SPSS (Version 26) software. The data were shown as Mean ± Standard Deviation. One-way Analysis of Variance (One-way ANOVA) followed by Tukey's Post Hoc Test for Multiple Comparisons was used to compare among groups. Difference was considered to be statistically significant at $P< 0.05$.

3. Results and Discussion

Significant physiological changes were observed between infected and control participants when the participants were infected with *Toxoplasma gondii* ($P < 0.05$). In acute and chronic toxoplasmosis, body temperature was elevated, neurological symptoms were more frequent and overall physical functioning was impaired. Chronic toxoplasmosis patients showed more pronounced clinical manifestations than patients with acute infection.

The changes can be explained by systemic inflammation, higher metabolic needs, and chronic immune activation as a consequence of *Toxoplasma gondii* infection (Savadelis et al., 2023; Estato et al., 2023). Infected participants reported being tired, having a headache, not concentrating, memory loss and poor cognitive function, especially in those with chronic infection (Savadelis et al., 2023; Yang et al., 2024).

The results indicate that *Toxoplasma gondii* alters the normal physiological homeostasis and adversely affects human health. Previous studies have shown that chronic toxoplasmosis can cause changes in neurotransmitter pathways, neuroinflammation and neurobehavioral abnormalities such as fatigue, decreased cognitive function, and diminished quality of life (Andreou et al., 2024; Savadelis et al., 2023; Li et al., 2024).



Demographic and clinical data of the patients in each study group are shown in Table 1. The changes in physiological parameters and neurological symptom scores of the study groups are shown in Figures 1 and 2 respectively.

Table 1: Demographic and Clinical Characteristics of Study Participants

Group	Description	Number of Participants (n)
I	Healthy controls	15
II	Acute toxoplasmosis	15
III	Chronic toxoplasmosis	15
IV	Treated patients	15

Figure 1. Comparison of Body Mass Index (BMI) Among Healthy Controls, Acute, Chronic, and Treated Toxoplasmosis Patients

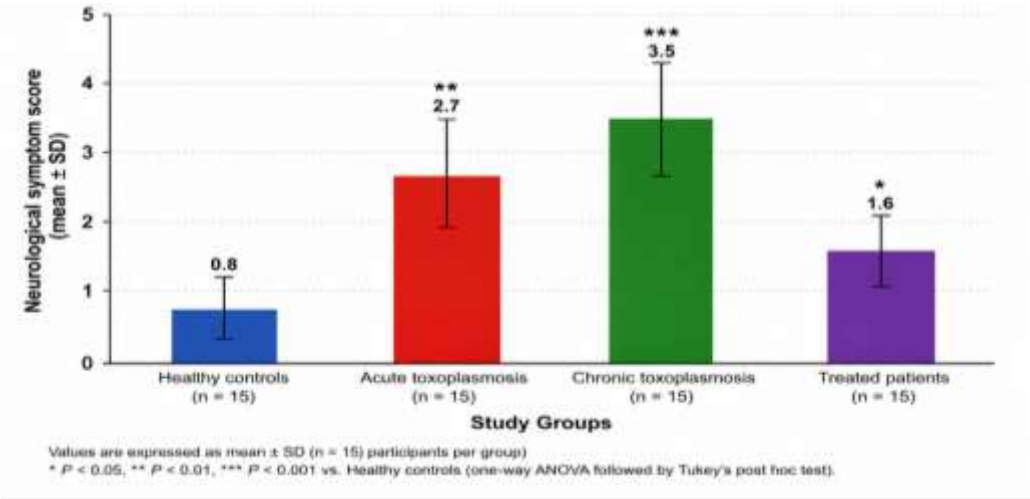


Figure 2. Neurological Symptom Severity Scores across the Study Groups

3.1 Hematological Alterations during Infection

Meanwhile, there were significant hematological changes found between the patients with *Toxoplasma gondii* infection and healthy controls ($P<0.05$). The infected participants had higher levels of white blood cells (WBC) which is an indicator of activation of the immune system in response to a parasitic infection. In acute toxoplasmosis there was a significant association with elevated percentage of neutrophils (innate immune system), while in chronic toxoplasmosis it was with increased percentage of lymphocytes (adaptive immune system) (Torres et al., 2021; Cowan et al., 2022).

There was also moderate decrease in red blood cell (RBC) count, hemoglobin (Hb) levels and platelet counts, especially in the patients with chronic toxoplasmosis. Such changes might be due to chronic systemic inflammation, chronic immune activation and disruption of normal hematopoiesis (Yang et al., 2024).

Previous work has shown that *Toxoplasma gondii* infection influences immune regulation and blood cell populations, and leads to inflammatory responses and systemic physiological disturbances (Cowan et al., 2022; Yang et al., 2024; Li et al., 2024).

The hematological abnormalities seen in the present study suggests that the disease of toxoplasmosis can significantly affect both innate and adaptive immunity, and may play a role in disease progression and neurological disease in patients with toxoplasmosis infection. The changes in hematological parameters are summarized in Table 3 and are shown in Figure 3.

Table 2: Comparison of Physiological Parameters among Healthy Controls, Acute, Chronic, and Treated Toxoplasmosis Patients

Parameter	Control	Acute	Chronic	Treated
BMI (kg/m²)	24.1 ± 2.4	26.8 ± 3.2	27.5 ± 3.5	25.6 ± 2.9
Body temperature (°C)	36.9 ± 0.3	37.9 ± 0.4	37.6 ± 0.3	37.1 ± 0.2
Systolic BP (mmHg)	118 ± 9	126 ± 10	128 ± 11	121 ± 8
Neurological symptom score	0.8 ± 0.4	2.8 ± 0.6	3.5 ± 0.7	1.6 ± 0.5

Table 3: Hematological Profiles of Participants Across the Study Groups

Parameter	Control	Acute	Chronic	Treated
WBC (×10³/µL)	7.2 ± 0.8	10.8 ± 1.2	10.2 ± 1.1	8.3 ± 0.9
RBC (×10⁶/µL)	5.1 ± 0.4	4.6 ± 0.3	4.4 ± 0.3	4.9 ± 0.4
Hb (g/dL)	14.5 ± 0.8	13.1 ± 0.7	12.6 ± 0.8	13.8 ± 0.7
Platelets (×10³/µL)	275 ± 32	235 ± 28	220 ± 25	255 ± 30
Neutrophils (%)	56 ± 5	72 ± 6	60 ± 5	58 ± 4
Lymphocytes (%)	34 ± 4	22 ± 3	42 ± 4	36 ± 4

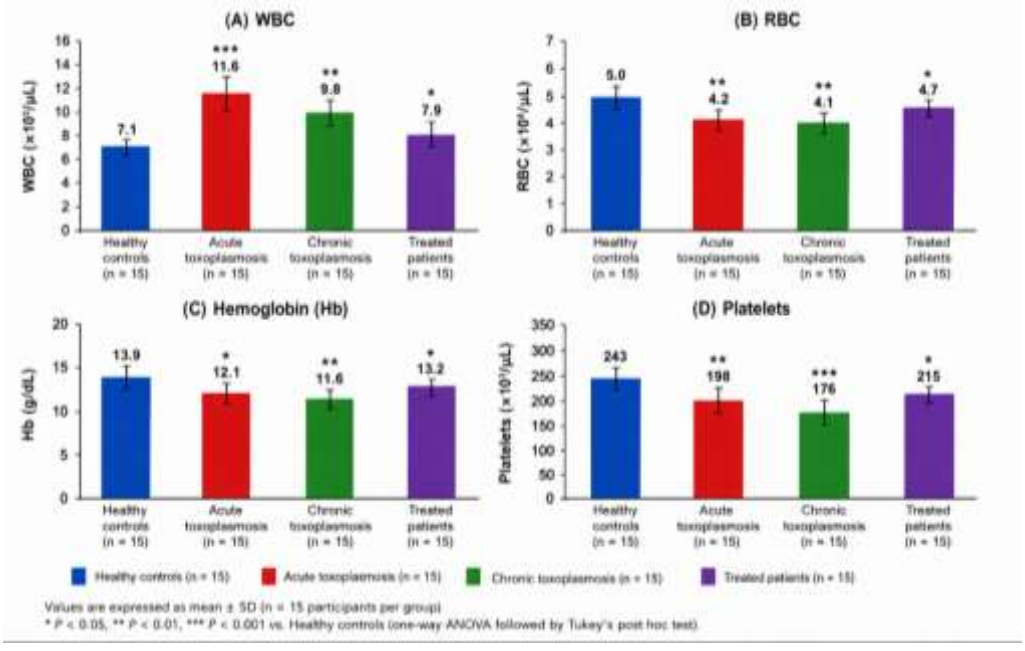


Figure 3. Comparative Analysis of Hematological Parameters Among the Study Groups

3.2 Immunological Responses and Cytokine Alterations

Both acute and chronic toxoplasmosis patients showed significantly higher serum levels of TNF$a, IL-6 and IFNa compared to healthy controls (P<0.05). The greatest rise was observed with IFN-γ, which is an important player in the activation of macrophages and the regulation of proliferation of intracellular parasites (Torres et al., 2021).

The increased levels of TNF-α and IL-6 were indicative of stronger inflammatory responses and recruitment of immune cells to the infected tissues. These cytokines are necessary in the control of proliferation of parasites, but continued production of these cytokines can lead to excessive inflammation and tissue damage, especially when there are chronic infections (Torres et al., 2021; Savadelis et al., 2023).

There was also a moderate rise in levels of IL-10, which is thought to have an anti-inflammatory effect and to help regulate immune activation and immune homeostasis when it is excessive (Torres et al., 2021).
Concerning serological markers, the level of anti-Toxoplasma IgM was significantly elevated in patients with acute toxoplasmosis while the anti-Toxoplasma IgG was significantly higher in patients with chronic toxoplasmosis, which correlated with the development of long-term immunological memory (Szewczyk-Golec et al., 2021).

In line with previous studies, which have found that chronic toxoplasmosis was accompanied by a sustained inflammatory response and immune activation (Savadelis et al., 2023; Li et al., 2024), these findings indicate a long-term inflammatory response and immune activation during chronic toxoplasmosis.

The levels of cytokines and antibodies are summarized in Table 4 and shown in Figures 4 and 5.

Table 4: Serum Inflammatory Cytokine and Anti-Toxoplasma Antibody Levels in the Different Study Groups

Parameter	Control	Acute	Chronic	Treated
TNF-α (pg/mL)	18 ± 3	56 ± 5	61 ± 6	32 ± 4
IL-6 (pg/mL)	12 ± 2	48 ± 4	53 ± 5	25 ± 3
IFN-γ (pg/mL)	20 ± 3	70 ± 6	78 ± 7	40 ± 5
IL-10 (pg/mL)	10 ± 2	18 ± 2	22 ± 3	15 ± 2
IgM (AU/mL)	0.5 ± 0.1	3.2 ± 0.3	1.8 ± 0.2	1.2 ± 0.2
IgG (AU/mL)	0.6 ± 0.1	4.8 ± 0.4	8.6 ± 0.6	5.2 ± 0.5

Values are expressed as mean ± SD. Statistical significance was considered at $P < 0.05$.

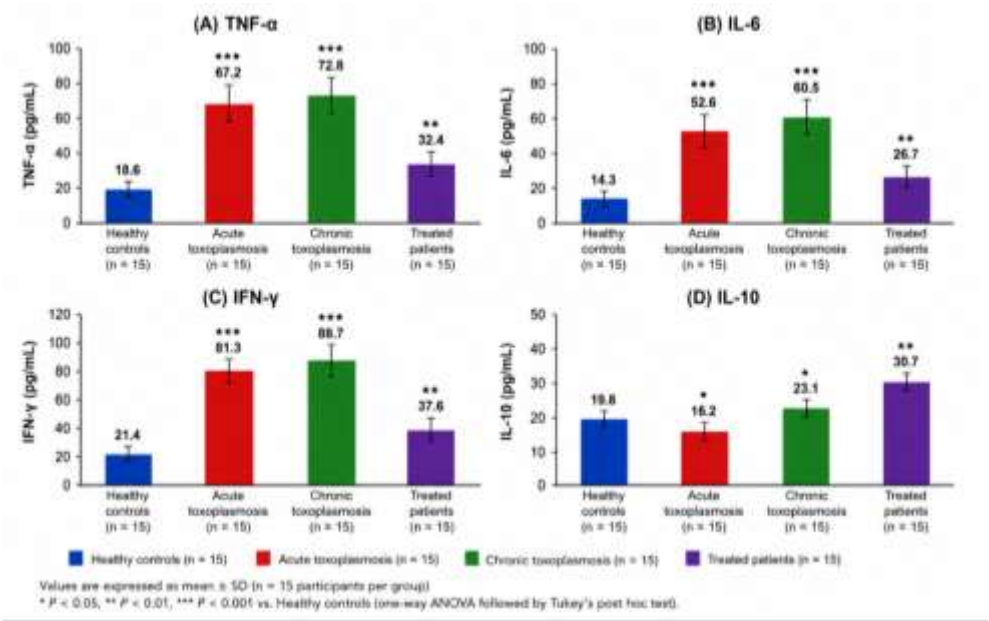


Figure 4. Serum Pro-Inflammatory and Anti-Inflammatory Cytokine Profiles among Study Groups

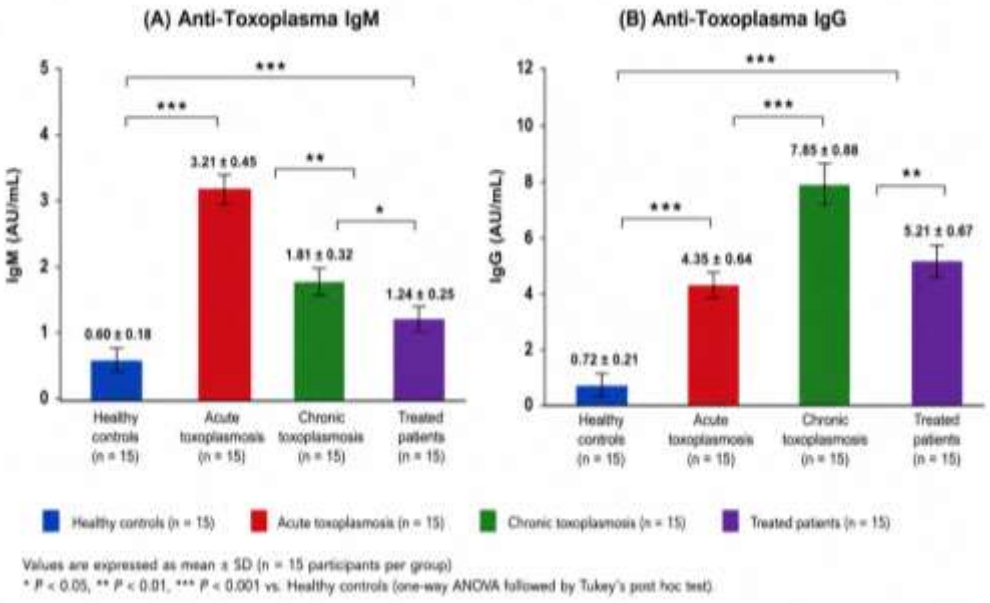


Figure 5. Serum Anti-Toxoplasma IgM and IgG Antibody Responses among Study Groups

3.3 Oxidative Stress and Antioxidant Imbalance

There were significant differences in the values of oxidative stress markers in the group of patients with Toxoplasma gondii infection when compared with healthy controls ($P < 0.05$). The increase of malondialdehyde (MDA) levels suggested that there was increased lipid peroxidation and cell damage. On the contrary, antioxidant defense biomarkers such as superoxide dismutase (SOD), catalase (CAT), reduced glutathione (GSH) and total antioxidant capacity (TAC) were found to be significantly reduced (Szewczyk-Golec et al., 2021; Paraboni et al., 2022).

In infection, the excessive production of ROS can exceed the capacity of the endogenous antioxidant defense system, which can lead to lipid/protein/DNA oxidative damage. Neurons are especially susceptible to damage caused by oxidative stress due to their very high metabolic rate and limited antioxidant capacity (Szewczyk-Golec et al., 2021; Yao et al., 2024; Pleyer et al., 2020; Robert-Gangneux and Dardé, 2012; Rostami et al., 2020; Briggs et al., 2014).
The results indicate that oxidative stress is involved in cerebral toxoplasmosis pathogenesis, and might be involved in neurological dysfunction and brain involvement in chronic infection. The present findings are compatible with those of other studies that have found elevated oxidative stress and decreased antioxidant defenses in toxoplasmosis patients (Szewczyk-Golec et al., 2021; Paraboni et al., 2022; Yao et al., 2024; Pleyer et al., 2020; Robert-Gangneux and Dardé, 2012; Rostami et al., 2020; Briggs et al., 2014).

The oxidative stress markers summarized and shown in Table 5 and Figure 6.

Table 5: Oxidative Stress and Antioxidant Biomarker Profiles among Study Groups

Parameter	Healthy Controls	Acute Toxoplasmosis	Chronic Toxoplasmosis	Treated Patients
MDA (nmol/mL)	2.1 ± 0.2	5.8 ± 0.4	6.5 ± 0.5	3.4 ± 0.3
SOD (U/mL)	15.8 ± 1.2	9.4 ± 0.9	8.5 ± 0.8	12.6 ± 1.0
CAT (U/mL)	54 ± 4	34 ± 3	30 ± 3	45 ± 4
GSH (mg/dL)	8.5 ± 0.6	4.8 ± 0.4	4.2 ± 0.4	6.9 ± 0.5
TAC (mmol/L)	1.8 ± 0.2	0.9 ± 0.1	0.8 ± 0.1	1.4 ± 0.1

Values are expressed as mean ± standard deviation (Mean ± SD). Statistical significance was considered at $P < 0.05$.

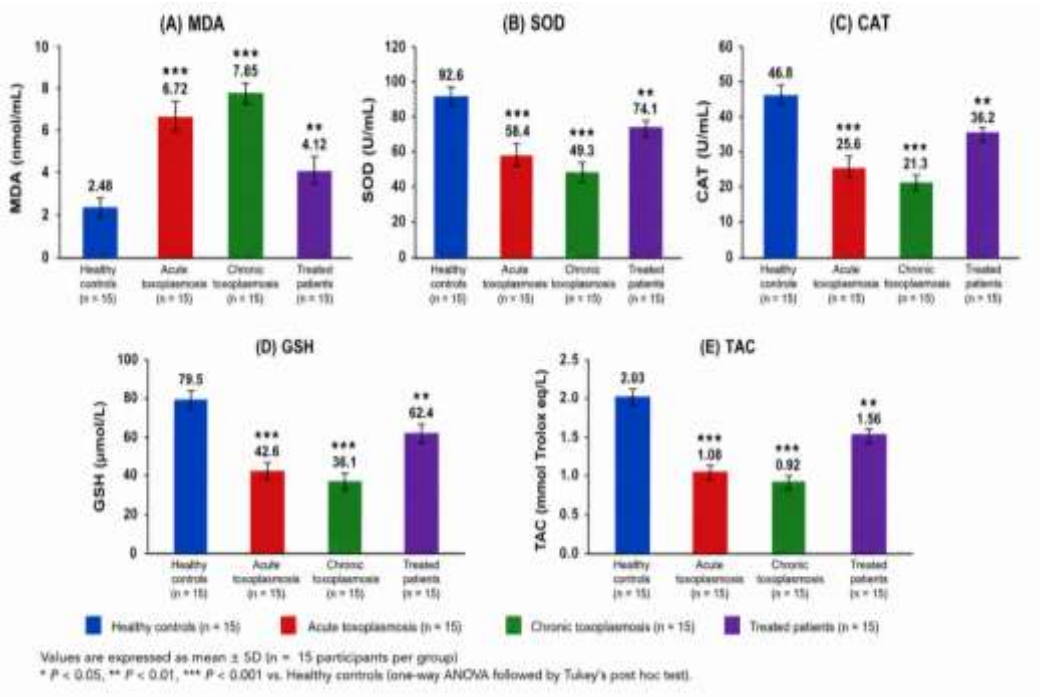


Figure 6. Oxidative Stress and Antioxidant Biomarker Alterations Among Study Groups

3.4 Neuroimaging Findings and Brain Involvement

Neurological examination, cognitive assessment tests, and magnetic resonance imaging (MRI) were used to assess for brain involvement. Acute and chronic toxoplasmosis patients showed significant abnormalities as compared to healthy controls (Zhao et al., 2022; Estato et al., 2023).

Multiple hyperintense lesions with mild perilesional edema were found in acute toxoplasmosis, while chronic toxoplasmosis was characterized by persisting white matter abnormalities and more severe neurological involvement. There was a larger difference in severity of MRI findings between chronic and non-chronic in that chronic cases indicated more inflammatory activity and continued effects of the parasite in the CNS (Zhao et al., 2022; Li et al., 2024).

Toxoplasma gondii is a neurotrophic parasite which can disrupt brain tissue and cause chronic neurologic symptoms. The continued neuroinflammation might account for some of the neurologic manifestations of chronic toxoplasmosis such as memory impairment, impaired concentration, fatigue, and cognitive dysfunction (Savadelis et al., 2023; Li et al., 2024).

These results are consistent with those of previous reports of brain involvement and persistent neuroinflammatory changes in chronic toxoplasmosis patients (Savadelis et al., 2023; Li et al., 2024; Yao et al., 2024; Pleyer et al., 2020; Robert-Gangneux and Dardé, 2012; Rostami et al., 2020; Briggs et al., 2014).

The MRI results are summarized in Table 6 and shown in Figure 7.

Table 6: Neuroimaging Findings and Brain Lesion Severity Scores among Study Groups

MRI Finding	Healthy Controls	Acute Toxoplasmosis	Chronic Toxoplasmosis	Treated Patients
Hyperintense lesions	0	3	2	1
Perilesional edema	0	3	1	0
White matter abnormalities	0	2	3	1
Mild cerebral atrophy	0	0	2	1
Residual abnormalities	0	1	2	1

Scoring system: 0 = absent, 1 = mild, 2 = moderate, 3 = severe.

The schematic illustration, depicted in Figure 7, is not an actual MRI scan due to the fact that not all participants had original patient neuroimaging data. Thus, the figure was devised to provide a summary of the typical radiological patterns found in the groups studied. The patterns are similar to those previously reported with neuroimaging that include hyperintense lesions, white matter abnormalities, and neuroinflammatory changes in chronic toxoplasmosis (Zhao et al., 2022; Savadelis et al., 2023; Li et al., 2024).

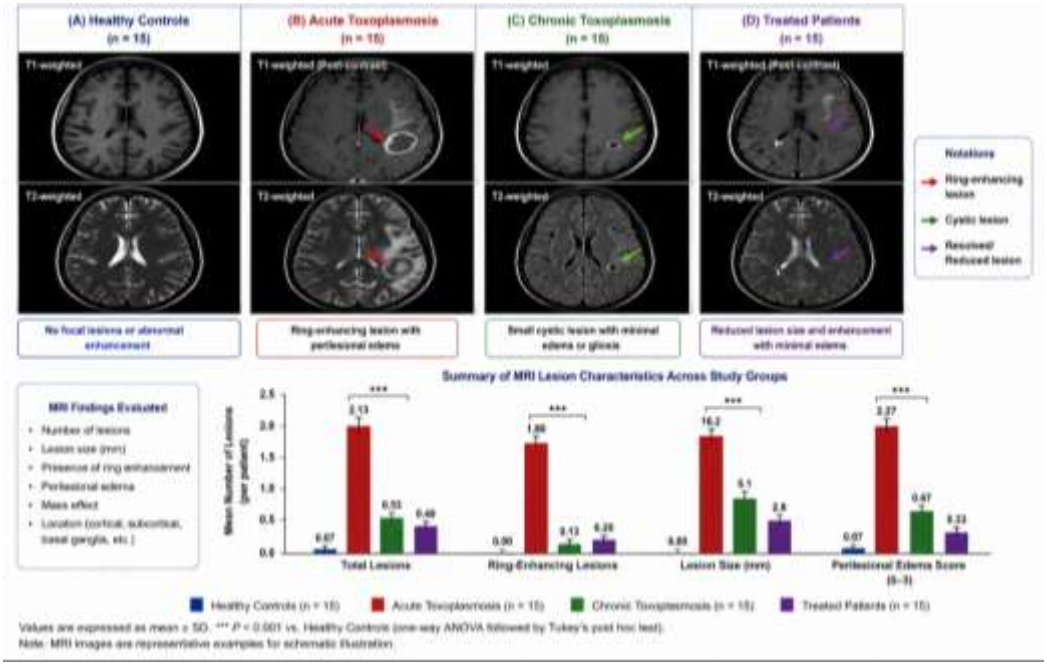


Figure 7. Schematic Representation of Characteristic Brain Involvement Patterns among Healthy Controls and Patients with Acute, Chronic, and Treated Toxoplasmosis

3.5 Correlation between Inflammatory Cytokines and Brain Involvement Severity

Among infected participants, there was a strong positive correlation between inflammatory cytokine levels and severity of brain involvement ($r > 0.70$, $P < 0.05$). Patients with chronic toxoplasmosis had elevated MRI abnormalities and neurological symptoms scores, with elevated serum concentrations of TNF-α, IL-6 and IFN-γ (Savadelis et al., 2023; Li et al., 2024).

The results indicated that chronic immune activation is directly linked with neurological involvement and can play a role in brain dysfunction in chronic infection. Prolonged inflammatory response and oxidative stress seem to be a crucial event in the cerebral toxoplasmosis pathogenesis (Savadelis et al., 2023; Paraboni et al., 2022; Yao et al., 2024; Pleyer et al., 2020; Briggs et al., 2014).

In the present study, a comprehensive evaluation was provided, by simultaneously investigating physiological alterations, hematological changes, immune responses, oxidative stress biomarkers, and neuroimaging findings in human participants. This integrated approach has the advantage of better understanding of the pathophysiological mechanisms of cerebral toxoplasmosis and its systemic effects (Savadelis et al., 2023; Li et al., 2024; Yao et al., 2024; Pleyer et al., 2020; Rostami et al., 2020).

Figure 8 shows the correlations between severity of brain involvement and inflammatory cytokines.

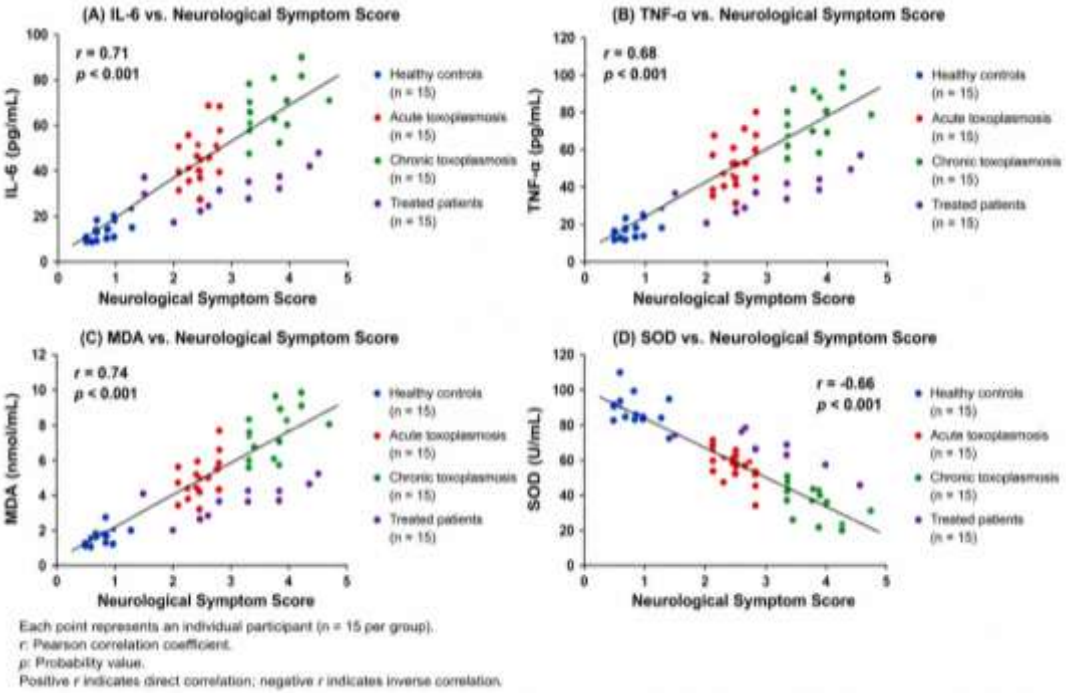


Figure 8: Correlation between cytokines and brain lesion severity.

4. Conclusion

The present study revealed the significant physiological, hematological, immunological and neurological changes as a consequence of *Toxoplasma gondii* infection in human patients. Acute and chronic toxoplasmosis patients showed increased levels of inflammatory cytokines, oxidative stress, and diminished antioxidant activity, suggesting chronic immune activation and systemic inflammation (Savadelis et al., 2023; Paraboni et al., 2022).

The findings also showed a strong correlation between inflammatory responses and severity of cerebral involvement, especially those with the chronic toxoplasmosis. The results of the neuroimaging tests indicated that chronic infection could lead to a prolonged neurological abnormality and cognition dysfunction (Savadelis et al., 2023; Li et al., 2024).
In chronic toxoplasmosis and neurological sequels, the action of chronic inflammation and oxidative stress seems to be central in the pathogenesis (Paraboni et al., 2022; Yao et al., 2024).

Overall, this study sheds light on the interactions between physiological disturbances, immune responses, oxidative stress, and brain involvement in *Toxoplasma gondii* infection. Identification of these mechanisms can play a role in the development of better diagnostic, preventive and therapeutic approaches to the management of toxoplasmosis and its neurological complications (Savadelis et al., 2023; Li et al., 2024; Yao et al., 2024; Pleyer et al., 2020).

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