



Article

Comparative Evaluation of Hematological Parameters in Renal Failure Patients with and Without Toxoplasma Gondii Infection

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Abstract: Background and Objective: Toxoplasmosis, caused by the widespread zoonotic parasite *Toxoplasma gondii*, may represent an important opportunistic infection in patients with chronic kidney disease, particularly those undergoing hemodialysis. Renal dysfunction is associated with uremia, chronic inflammation, and immune disturbances that may increase susceptibility to infectious diseases. As hematological parameters are important indicators of the health status of patients with renal failure, this study aimed to evaluate and compare hematological parameters in renal failure patients with and without *T. gondii* infection and in a healthy control group. Methods: A total of 108 blood samples were analyzed, including 68 samples from patients diagnosed with renal failure and undergoing hemodialysis and 40 samples from apparently healthy volunteers serving as controls. *T. gondii* IgG and IgM antibodies were detected using commercial ELISA kits. Complete blood count (CBC) parameters and erythrocyte sedimentation rate (ESR) were determined for all participants. Results: The seroprevalence of chronic *T. gondii* infection, indicated by IgG positivity, was significantly higher among renal failure patients (32.35%, 22/68) than among controls (12.5%, 5/40) ($P < 0.05$). Acute infection, indicated by IgM positivity, was detected in 4.41% (3/68) of renal failure patients compared with 0% of controls, with no statistically significant difference. Both *T. gondii*-infected and uninfected renal failure patients showed significantly lower HGB, HCT, and MCHC levels than healthy controls ($P < 0.01$). WBC counts were significantly lower in renal failure patients than in controls ($P < 0.05$), while LYM% was significantly reduced and ESR was significantly elevated ($P < 0.01$). MCH also differed significantly among the three groups ($P < 0.05$), whereas MCV, PLT, NEU%, MONO%, EOS%, and BASO% showed no significant differences. No significant differences were observed between *T. gondii*-infected and uninfected renal failure patients for most hematological parameters. Conclusion: Renal failure was associated with significant hematological alterations, including reduced HGB, HCT, MCHC, WBC, and LYM%, and elevated ESR. Although some parameters differed between *T. gondii*-infected and uninfected patients, renal dysfunction itself appeared to be the major driver of these abnormalities. Further studies are warranted to clarify the potential contribution of *T. gondii* infection to hematological changes in renal failure patients.

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Keywords: Toxoplasmosis; *Toxoplasma gondii*; renal failure; hemodialysis; hematological parameters.

1. Introduction

Protozoan parasites are responsible for numerous important and widely distributed diseases that affect millions of people worldwide. Among these parasitic diseases, toxoplasmosis is considered one of the most important zoonotic infections. It is caused by *Toxoplasma gondii*, an obligate intracellular protozoan parasite capable of infecting

humans, other mammals, and birds. *T. gondii* is one of the most widely distributed protozoan parasites worldwide and has a broad host range [1]-[4]. Felids, particularly domestic cats, are the definitive hosts of *T. gondii*, whereas humans and other warm-blooded animals serve as intermediate hosts. The life cycle of the parasite comprises three major infectious stages: tachyzoites, bradyzoites enclosed within tissue cysts, and sporozoites contained within oocysts [5]-[7]. Human infection occurs mainly through the ingestion of food or water contaminated with oocysts shed in the feces of infected cats, or through the consumption of raw or inadequately cooked meat containing tissue cysts. In addition, *T. gondii* can be transmitted congenitally from an infected mother to the fetus through the placenta and through blood transfusion and organ transplantation [7], [8]. The pathological effects of *Toxoplasma gondii* are generally limited in immunocompetent individuals. In contrast, in immunocompromised patients including those with immunodeficiency resulting from immunosuppressive therapy, acquired immunodeficiency syndrome (AIDS), autoimmune diseases, malignancies, or organ transplantation, the parasite may behave opportunistically. In such individuals, reactivation of latent infection may occur and progress to severe clinical manifestations. [9]-[11].

Chronic kidney disease (CKD) is a progressive disorder characterized by a persistent and irreversible decline in renal function, resulting in impaired filtration and accumulation of metabolic waste products. As the disease advances, particularly toward end-stage kidney disease, patients may develop various systemic complications, including metabolic disturbances, chronic inflammation, and cardiovascular disorders. In addition, renal dysfunction is associated with alterations in both cellular and humoral immune responses, which can increase susceptibility to infectious diseases. These immunological abnormalities are particularly evident in patients receiving hemodialysis [12]-[15]. Dialysis is a renal replacement therapy that assists in removing metabolic waste products and excess fluid from the blood in patients with severe renal dysfunction. Although it provides essential support when kidney function is inadequate, dialysis does not completely replicate the physiological functions of healthy kidneys. The process primarily depends on mechanisms such as diffusion and ultrafiltration to regulate the composition and volume of body fluids. In patients with compromised physiological and immune defenses, repeated exposure to dialysis may be associated with increased oxidative stress and other systemic effects [13], [16]. Patients with chronic kidney disease (CKD), particularly those undergoing hemodialysis (HD), may be at increased risk of *T. gondii* infection or reactivation. This susceptibility may be associated with impaired immune function resulting from uremia, chronic inflammation, and multiple comorbidities, which can increase vulnerability to opportunistic infections [17], [18]. Alterations in hematological parameters, such as red and white blood cell counts and platelet indices, are commonly observed in patients with chronic kidney disease (CKD) [19], [20].

The aim of the present study was to identify and compare variations in hematological parameters between renal failure patients infected with *Toxoplasma gondii* and those without infection.

2. Material and Methods

2.1-Samples Collection

A total of 108 blood samples were collected for this study, consisting of 68 samples from patients diagnosed with renal failure and 40 samples from apparently healthy volunteers, who served as the control group.

Venous blood samples were obtained from each participant via a standard venipuncture procedure using a tourniquet and a 10 mL sterile disposable syringe. Approximately 5-7 mL of blood was drawn and carefully divided into three types of collection tubes:

1. Two milliliters (2 mL) of blood were transferred into a pre-labeled EDTA tube containing an anticoagulant for hematological analyses.

2. One and a half milliliters (1.5 mL) of blood were placed into a sodium citrate tube containing an anticoagulant for erythrocyte sedimentation rate (ESR) determination.

3. The remaining blood was transferred into a plain (clot activator) tube and allowed to clot at room temperature for 15–30 minutes. Samples were then centrifuged at 3000 rpm for 10 minutes to separate the serum, which was carefully aliquoted into sterile Eppendorf tubes and stored at -20°C until used for serological assays.

2.2-Laboratory Tests

Laboratory investigations involved the determination of hematological and serological parameters for all studied groups. *Toxoplasma gondii* antibodies were detected using BioCheck ELISA kits (both IgM and IgG), strictly following the manufacturer's instructions. Complete blood count (CBC) analyses were performed using the CELL-DYN Ruby hematology analyzer. Additionally, the Microsed System was employed to determine the erythrocyte sedimentation rate (ESR).

2.3-Statistical Analysis

The data were statistically analyzed using the Chi-square test and analysis of variance (ANOVA) with Minitab Statistical (17). Differences between means were further assessed using Duncan's Multiple Range Test (DMRT). Statistical significance was considered at $P < 0.05$ and $P < 0.01$.

3. Results

The results presented in Figure (1) show the percentages of infection among the samples studied using the ELISA test. The prevalence of chronic infection among renal failure patients reached 32.35% (22 cases), compared with 12.5% (5 cases) in the control group. Statistical analysis revealed a significant difference between the two groups at a probability level of $P < 0.05$. In contrast, the prevalence of acute infection among renal failure patients was 4.41% (3 cases), compared with 0% in the control group. Statistical analysis indicated no significant difference between the two groups.

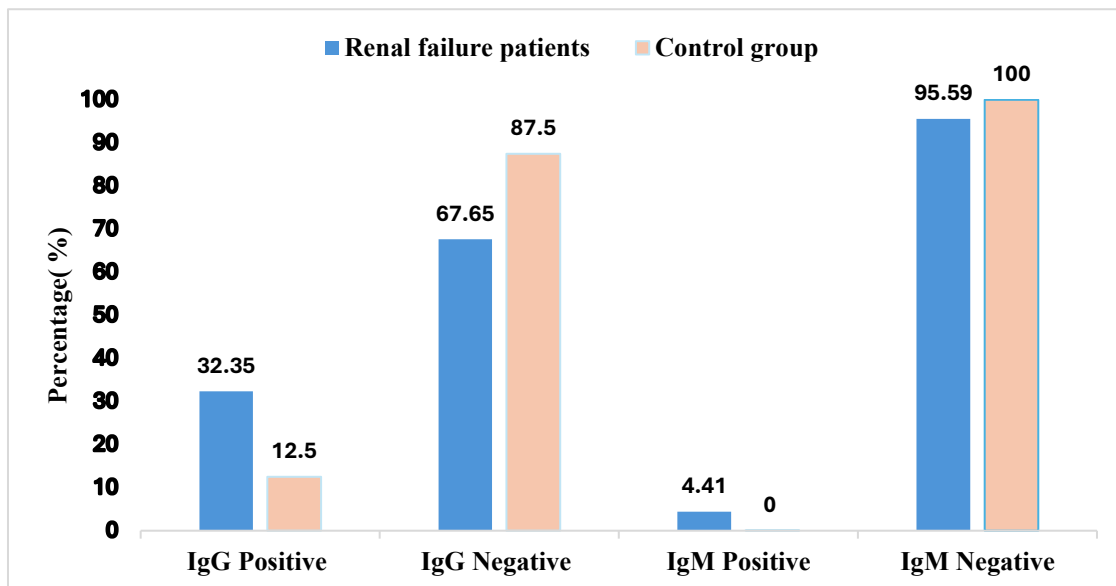


Figure 1. The percentage of renal failure patients infected with toxoplasmosis and control group according to ELISA test

The analysis revealed in Table (1) a significant reduction in hemoglobin (HGB) levels among both *T. gondii*-infected and uninfected renal failure patients compared with the control group ($P < 0.01$). Similarly, hematocrit (HCT) values were markedly lower in infected and uninfected patients than in controls with statistically significant differences among the three groups ($P < 0.01$). No significant differences were detected in mean corpuscular volume (MCV). Mean corpuscular hemoglobin (MCH) showed a statistically

significant difference among the studied groups ($P < 0.05$); the MCH level was lower in *T. gondii*-infected renal failure patients compared with the control group, while the uninfected renal failure group showed an intermediate level. Mean corpuscular hemoglobin concentration (MCHC) was significantly lower in both renal failure groups than in the control group ($P < 0.01$). Erythrocyte sedimentation rate (ESR) was significantly elevated in both infected and uninfected renal failure patients relative to controls ($P < 0.01$). Platelet count (PLT) showed no significant differences among the groups. For none of these parameters was a significant difference observed between *T. gondii*-infected and uninfected renal failure patients.

Table 1. Hematological variables in renal failure patients infected with Toxoplasmosis and uninfected compared with the control group

Hematological variables	Renal failure with toxoplasmosis	Renal failure without toxoplasmosis	Control group	Statistical analysis
HGB g/dL	9.362 ± 2.631	8.677 ± 1.746	13.510 ± 1.675	F=44.12**
HCT %	29.12 ± 7.31	29.277 ± 5.563	41.55 ± 7.65	P=0.00086
MCV fL	89.44 ± 7.34	90.52 ± 8.26	89.88 ± 7.58	F=42.78**
MCH Pg	28.584 ± 3.046	29.100 ± 3.252	30.44 ± 2.00	P=0.00081
MCHC g/dL	31.91 ± 1.69	32.11 ± 1.80	33.68 ± 1.35	F=0.14 ^{ns}
PLT 10 ³ /μL	222.57 ± 68.26	218.8 ± 146.9	238.2 ± 51.3	P=0.873
ESR mm/hr	37.99 ± 48.09	48.11 ± 35.95	7.115 ± 5.031	F=3.28*
				P=0.0428
				F=9.32**
				P=0.0002
				F=0.28 ^{ns}
				P=0.757
				F=15.92**
				P=0.00031
		Mean ± Standard deviation		

As illustrated in Table (2), the total white blood cell (WBC) count was lower in both renal failure groups, including patients infected and non-infected with *Toxoplasma gondii*, compared with the healthy control group ($P < 0.05$), with no significant difference between the two patient groups. Lymphocyte (LYM) percentages were significantly lower in all renal failure patients compared to the control group, with a high statistical significance of $P < 0.01$, whereas no significant difference was observed between *T. gondii*-infected and non-infected renal failure patients. For the remaining differential leukocyte types neutrophils (NEU), monocytes (MONO), eosinophils (EOS), and basophils (BASO) the statistical analysis indicated no significant differences ($P > 0.05$) among the three study groups.

Table 2. White Blood Cells count in renal failure patients infected and Non-infected with Toxoplasmosis compared with the control group

Hematological variables	Renal failure with toxoplasmosis	Renal failure without toxoplasmosis	Control group	Statistical analysis
WBC 10 ³ /μL	6.91 ± 1.25	6.42 ± 2.05	7.63 ± 1.41	F=3.17*
NEU %	58.09 ± 23.27	62.52 ± 32.21	51.36 ± 12.36	P=0.0487
LYM %	15.15 ± 5.23	13.72 ± 7.88	29.13 ± 8.66	F=1.10 ^{ns}
				P=0.337
				F=34.44**
				P=0.00062

MONO	5.488 ± 2.189	6.330 ± 2.825	5.296 ± 1.561	F=1.09 ^{ns}
%	A	A	A	P=0.340
EOS	3.129 ± 3.000	3.018 ± 2.371	2.967 ± 1.434	F=0.03 ^{ns}
%	A	A	A	P=0.969
BASO	0.9556 ± 0.4645	1.055 ± 1.563	0.9531 ± 0.3496	F=0.41 ^{ns}
%	A	A	A	P=0.665
Mean ± Standard deviation				

4. Discussions

The significant reductions in HGB and HCT observed in the present study are corroborated by [21], which reported a highly significant decrease in HGB levels in both infected and uninfected renal failure patients compared to healthy controls. Previous reports have demonstrated a high prevalence of severe anemia in hemodialysis and chronic kidney disease (CKD) [22]–[24]. These findings are in agreement with numerous previous studies that have identified anemia as a common hematological complication in chronic kidney disease (CKD). The markedly lower HCT values observed in both patient groups further confirm the presence of anemia associated with renal failure. Similar reductions in HGB and HCT have been reported by earlier investigations, which attributed these alterations to iron deficiency, chronic inflammation, and nutritional deficiencies frequently encountered in CKD patients. The lack of a statistically significant difference between *T. gondii*-infected and uninfected patients suggests that renal dysfunction itself plays a more substantial role in determining these parameters than parasitic infection alone. Nevertheless, some studies have suggested that chronic toxoplasmosis may worsen anemia through immune-mediated mechanisms and inflammatory cytokine release, which could partially explain the observed trends.

The reduction in MCH among renal failure patients may be associated with disturbances in erythropoiesis and iron metabolism commonly observed in renal dysfunction. Chronic inflammation may also interfere with iron utilization and hemoglobin synthesis, contributing to reduced MCH levels. The absence of a clear difference between *T. gondii*-infected and uninfected renal failure patients suggests that renal dysfunction and its associated hematological disturbances may have a greater influence on MCH than *T. gondii* infection alone. The significant reduction in mean corpuscular hemoglobin concentration (MCHC) among renal failure patients aligns with previous research demonstrating hypochromic features in CKD patients. This reduction may be attributed to altered iron metabolism, decreased intestinal iron absorption, and the inhibitory effect of inflammatory mediators on erythropoiesis. Studies investigating parasitic infections, including *T. gondii*, have also suggested a possible role of chronic infection in disrupting iron homeostasis, which may contribute to lower MCHC values [25].

The elevated ESR in renal failure patients is consistent with studies [22], [26] which similarly reported markedly elevated ESR levels in hemodialysis patients, representing chronic systemic inflammation associated with uremia. This finding is consistent with earlier studies reporting elevated ESR levels in CKD patients as a result of persistent inflammation and oxidative stress. Although *T. gondii* infection can stimulate the host immune response and contribute to inflammatory processes, the absence of a significant difference in ESR between infected and uninfected patients in the present study suggests that renal failure and its associated inflammatory state may have had a greater influence on ESR than toxoplasmosis.

Regarding platelet count, studies [23], [27] reported significantly reduced platelet counts in renal failure patients, and study [21] likewise reported a significant reduction (thrombocytopenia) in platelet count in patients compared to healthy controls; these reports disagree with the present finding of no significant differences among the groups. The discrepancy between the present findings and those of previous studies may be related to differences in disease severity, duration of renal failure, dialysis status, sample

size, or other clinical and demographic characteristics of the studied populations. Furthermore, the absence of a significant difference between *Toxoplasma gondii*-infected and uninfected patients suggests that *T. gondii* infection did not have a measurable additional effect on platelet count in the present study. Overall, the findings suggest that platelet abnormalities were not a prominent hematological feature in the studied renal failure patients.

The lower total WBC count in renal failure patients agrees with the results of study [21], which reported a significant decrease in WBC counts among both infected and uninfected renal failure patients compared with healthy controls. It is also consistent with study [23], which reported that a considerable proportion of patients with kidney failure admitted to the nephrology department had markedly reduced WBC counts. Similarly, studies [24] and [28] reported reductions in total WBC counts among hemodialysis patients and individuals with *Toxoplasma gondii* infection respectively.

The significant reduction in WBC counts observed in the present study may be associated primarily with the hematological and immune disturbances accompanying renal failure. Chronic renal dysfunction, particularly in patients undergoing hemodialysis, is associated with uremia, chronic inflammation, metabolic disturbances, and impaired immune regulation. These conditions may affect the production, distribution, survival, and functional activity of leukocytes. In addition, the hemodialysis procedure itself may contribute to changes in circulating leukocyte numbers. Therefore, the reduction in WBC counts in both infected and non-infected renal failure patients suggests that renal failure and its associated complications may have a greater influence on leukocyte counts than *T. gondii* infection alone.

The lower lymphocyte percentage agrees with study [24], which reported significantly lower lymphocyte percentages in hemodialysis patients compared with healthy individuals. The decreased lymphocyte percentage may reflect the immune dysfunction associated with chronic renal failure. Uremic conditions can impair cellular immunity and alter both the number and function of lymphocytes. Chronic inflammation, metabolic abnormalities, nutritional disturbances, and repeated exposure to hemodialysis may further contribute to alterations in lymphocyte homeostasis. In contrast, the present findings disagree with study [21], which reported no statistically significant difference in LYM% between renal failure patients and healthy controls. Although LYM% was significantly lower in renal failure patients compared with controls, the lack of a significant difference between *T. gondii*-infected and non-infected renal failure patients suggests that *T. gondii* infection did not produce an additional measurable effect on lymphocyte percentages in the patients studied. The predominant influence of renal failure-associated immune dysfunction may have masked any potential effect of chronic *Toxoplasma gondii* infection. Furthermore, most seropositive individuals identified by IgG antibodies may represent previous or latent infection rather than active disease and therefore may not exhibit substantial changes in circulating leukocyte populations.

The absence of significant differences in the remaining leukocyte subsets (NEU, MONO, EOS and BASO) suggests that these subsets were relatively stable across the studied groups. This may indicate that the hematological effect of renal failure in the present sample was more evident in the total WBC count and lymphocyte percentage than in the individual differential leukocyte populations. Likewise, the absence of significant differences between *Toxoplasma gondii*-infected and uninfected patients does not provide evidence of an additional measurable effect of *Toxoplasma gondii* infection on these parameters

5. Conclusion

The present study demonstrated that renal failure was associated with significant alterations in several hematological parameters, including reduced HGB, HCT, MCHC, WBC count, and LYM%, as well as increased ESR compared with healthy controls. MCH also showed a significant difference among the studied groups, whereas MCV, PLT, NEU%, MONO%, EOS%, and BASO% showed no significant differences.

Although *Toxoplasma gondii* infection was more prevalent among renal failure patients than healthy controls, no consistent significant differences in hematological parameters were observed between *T. gondii*-infected and uninfected renal failure patients. These findings suggest that renal dysfunction and its associated inflammatory and hematological disturbances may have a greater influence on the measured hematological parameters than *T. gondii* infection itself. Further studies involving larger sample sizes and additional immunological and biochemical parameters are recommended to clarify the potential contribution of *T. gondii* infection to hematological alterations in patients with renal failure.

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