Full Length Research Article
Advancements in Life Sciences – International Quarterly Journal of Biological Sciences

ARTICLE INFO

Date Received: 23/06/2023;
Date Revised: 04/09/2023;
Date Published Online: 20/10/2023;
Dated Updated: 04/09/2025

Authors' Affiliation:
Department of Biology, College of Education for Pure Science (Ibn Al- Haitham), University of Baghdad, Baghdad - Iraq

***Corresponding Author:**
Sarah Ali Saeed
Email: sarah.a.saeed91@gmail.com

How to Cite:
Saeed SA, Al-Aubaidi IK (2023). Assessment of Interleukin-10 Levels in Iraqi Diabetic Type 2 Patients Infected with Toxoplasmosis. Adv. Life Sci. 10S(1): 62-66.

Keywords:
Toxoplasmosis; DMT2; IgM; IgG; IL-10

Editorial Note:
This article has been updated with language corrections.

Open Access

Assessment of Interleukin-10 Levels in Iraqi Diabetic Type 2 Patients Infected with Toxoplasmosis

Sarah Ali Saeed*, Israa Kasim Al-Aubaidi

Abstract

Background: Toxoplasmosis is a disease caused by the obligate intracellular parasite Toxoplasma gondii, which infects birds and mammals as intermediate hosts and felids as definitive hosts. Diabetes mellitus is a global metabolic disorder characterized by hyperglycemia. Interleukin-10 is an immunoregulatory cytokine that plays a main role in modulating inflammation, it's considered as main inhibitory cytokine against the action of inflammatory cytokines such as IL-12. The main aim of this study is to assess the levels of IL-10 in Iraqi diabetic type 2 infected with toxoplasmosis.

Methods: This study included 109 Iraqi patients with type 2 diabetes and 80 healthy individuals. Samples were collected from a private laboratory in Baghdad, Iraq, from March to June 2022. The mean age of participants was 49.9 ± 1.29 years.

Results: The diabetic group had significantly higher glycemic markers, with a mean HbA1c of 7.9 ± 0.178%, fasting blood sugar (FBS) of 174.55 ± 3.96 mg/dl, and random blood sugar (RBS) of 216.89 ± 4.96 mg/dl. A total of 51/109 (46.8%) diabetic patients were seropositive for anti-*Toxoplasma* IgG (mean: 34.95 ± 7.5 UI/mL). Among the non-diabetic cohort, 30 individuals (classified as the toxoplasmosis-positive control group) were also seropositive (mean: 32.7 ± 8.45 UI/mL). While, all samples were seronegative for IgM anti-*Toxoplasma*. The group of healthy control has the highest levels of IL-10 in ELISA 320.43 ± 17.64 pg/ml followed by the group of diabetic patients 138.38 ± 5.69 pg/ml. Also, the concentration of this interleukin was in the group of toxoplasmosis patients which considered as a control positive 115.45 ± 4.44 pg/ml. While, the group of diabetic patients with toxoplasmosis has the lowest concentration of the interleukin 102.3 ± 7.05 pg/ml with highly significant differences.

Conclusion: This study found that IL-10 levels were highest in the healthy control group compared to all other study groups.

Introduction

Toxoplasmosis is a widespread parasitic zoonotic disease caused by *Toxoplasma gondii* and occurs worldwide. It has a worldwide distribution and is one of the most widespread sources of infection in Iraq. *T. gondii* is an obligate intracellular protozoan parasite that infects almost all warm-blooded animals, with mammals such as birds and humans serving as intermediate hosts. The final host of this parasite is the cat [1, 2]. Infection occurs mainly by eating food and drinking water contaminated with *T. gondii* oocysts or tissue cysts. Toxoplasmosis is an infectious disease of widespread concern, but the disease has no unique clinical manifestations. *T. gondii* is an opportunistic parasite that infects immunocompromised individuals [3-5].

This parasite efficiently spreads to all organs of the body due to its ability to escape into macrophages and dendritic cells. Eventually, immune compression against the parasite leads to the formation of cysts containing slow-growing bradyzoites [3]. Toxoplasmosis can cause fetal death if the parasite is transmitted through the placenta (transplacentally) or during vaginal delivery from an infected mother [6].

Diabetes mellitus (DM) is a syndrome caused by abnormal metabolism accompanied by inappropriate hyperglycemia due to an absolute deficiency of insulin secretion or decreased bioavailability of insulin, or both. Diabetic patients are prone to develop systemic microangiopathy, atherosclerosis, and neuropathy. Currently, the number of cases of this disease is increasing in developed countries around the world, which can adversely affect the quality of life of people with diabetes, making it a global disease problem [7, 8]. It is now very clear that diabetes is one of the chronic non-communicable diseases that can affect the entire world population in both developed and non-developed countries [9]. Diabetes is mainly classified into two types: Type 1 Diabetes Mellitus (T1DM), which is insulin-dependent, and Type 2 Diabetes Mellitus (T2DM), which is non-insulin-dependent. T2DM is characterized by abnormally high blood sugar levels caused by a relative deficiency of insulin [10, 11].

T. gondii can infect nucleated cells such as pancreatic cells, causing β-cell destruction and insulin secretion. In addition, the risk of acute and chronic pancreatitis and diabetes is also increased [12]. Bradyzoites have been identified in tissue cysts, bile duct epithelial cells, and acinar cells of pancreatic tissue [13, 14]. However, T2DM is thought to be a chronic inflammatory disease that causes multiple alterations in immune cell function [15, 16, 17]. *T. gondii* induces one of the most potent innate proinflammatory responses of any infectious agent. In addition, it manipulates immune responses specific to immunization against *T. gondii*. A delicate balance exists between parasite and host, including a chain of formatted cellular interactions involving enterocytes, dendritic cells, neutrophils, natural killer cells and macrophages [18, 19].

The anti-inflammatory cytokine IL-10 plays an important role in limiting the detrimental pathological effects of the inflammatory response in toxoplasmosis. This cytokine is secreted by macrophages, dendritic cells, B cells, Th2 cells, and regulatory T cells [20]. Suppression of the T cell-dependent immune system by IL-10 is primarily aimed at preventing overwhelming inflammation that ultimately leads to death. In addition, IL-10 inactivates macrophages and IFN-gamma strongly stimulates *Toxoplasma* acidity, promoting parasite intracellular survival. Consequently, IL-10 stimulates her *T. gondii* post-infection suppression, is beneficial to both parasite and host, and promotes a consistent host-parasite relationship [21].

The aim of this study is to find the role of IL-10 and assess its levels in Iraqi diabetic type 2 patients infected with toxoplasmosis in comparison with other studied groups.

Methods

Subjects and Samples

The current study included 189 participants, who were initially divided into two main cohorts based on their diabetic status: 109 patients with T2DM and 80 non-diabetic individuals. 109 samples from Iraqi type 2 diabetic patients who were diagnosed by experts and participated in diabetes testing at private laboratories in Baghdad, Iraq between March and June 2022. Eighty Iraqi specimens collected from non-diabetic patients during the period were compared by age. The age of participants ranged from 15 to 85 years. Five ml of venous blood was drawn from each sample. Serum was then separated in gel tubes and used for diabetes, toxoplasmosis assays, and IL-10 concentration measurements.

Diabetes mellitus diagnosis

Blood glucose measurement by fasting test followed by random testing using the Glucose Architect Kit (Abbott GmbH, Germany) and measurement of glycated hemoglobin levels using the Hemoglobin A1C Architect Kit (Abbott GmbH, Germany) according to the manufacturer's instructions.

T. gondii diagnosis

Toxoplasma antibody IgM and IgG Chemiluminescent Microparticle Immunoassay (CMIA) Architect Toxo IgM/G Kit (Abbott GmbH, Germany) was used to detect *T. gondii* according to the manufacturer's instructions.

Assessment IL-10 levels

IL-10 (interleukin 10) levels were measured via using a sandwich enzyme-linked immunosorbent assay (ELISA) kit for human IL-10 (mybiosource Inc., USA) according to the manufacturer's protocol.

Statistical Analysis

The Statistical Analysis System (SAS, version 22) was used for data analysis. A program that assesses the impact of various factors on research parameters. Analysis of variance (ANOVA) followed by the least significant difference (LSD) test was used to compare means between groups. The study used a chi-square test to make a significant comparison between percentages with probabilities 0.05 and 0.01.

Results

Diabetes mellitus diagnosis

As demonstrated in Table 1, the diabetic group had significantly higher glycemic markers than the healthy control group. The difference was significant for HbA1c ($p \leqslant 0.05$) and highly significant for fasting and random blood sugar tests ($p \leqslant 0.01$). The test shows a highly significant difference ($P \leqslant 0.01$) in random test and glycated hemoglobin test.

T. gondii diagnosis

According to the chemiluminescent microparticle immunoassay revealed in table (2), 51/109 of diabetic patients had the highest anti-*Toxoplasma* IgG level at 34.95 ± 7.5 UI/ml followed by 30/80 of non-diabetic controls at 32.7 ± 8.45 UI/ml with significant difference, the study groups were divided into the following four groups according to their anti-*Toxoplasma* IgG values. diabetic patients infected with toxoplasmosis, diabetic patients only, non-diabetic individuals infected with toxoplasmosis considered as a positive control and healthy individuals are considered as a negative control. However, all samples from diabetic and non-diabetic controls were seronegative for anti-*Toxoplasma* IgM and were significantly different.

Assessment of IL-10

From the total cohort, 30 samples from each of the four subgroups were selected for IL-10 analysis using a sandwich ELISA, which is demonstrated in table (3).

Groups	Total No. of samples for each group	Mean ± SE of HbA1C (Glycated Hemoglobin)	Upper Value	Lower Value	Mean ± SE of FBS mg/dl (Fasting blood sugar)	Upper Value	Lower Value	Mean ± SE of RBS mg/dl (Random blood sugar)	Upper Value	Lower Value
Diabetic Patients	109	7.9 ± 0.178	15.5	5.3	174.55 ± 3.96	300	120	216.89 ± 4.96	410	125
Non-Diabetic Control	80	4.98 ± 0.044	5.4	4.3	96.65 ± 0.749	98	81	160.25 ± 2.69	195	109
LSD value		1.667 *			26.381 **			31.093 **		
P-value		0.0252			0.0063			0.0058		
Significant * (P≤0.05), Highly significant ** (P≤0.01).										
Reference range of HbA1C Normal < 5.7 Prediabetes 5.7 – 6.4 Diabetes ⩾ 6.5.		**Reference range of FBS** Normal < 100 mg/dl. Prediabetes 101 – 125 mg/dl. Diabetes ⩾ 126.								
Reference range of RBS: over 200 mg/dl after two hours refers to diabetes.										

Table 1: HbA1C, FBS and RBS concentrations in the diabetic and non-diabetic groups of the diabetic tests

Groups	Total No. of samples for each group	Mean ± SE of Toxo IgG UI/mL	Upper Value	Lower Value	Mean ± SE of Toxo IgM UI/mL	Upper Value	Lower Value
Diabetic patients with toxoplasmosis	51 (26.98%)	34.95 ± 7.5 a	217	0.6	0.082 ± 0.0052 a	0.2	0.02
Diabetic patients	58 (30.69%)	0.024 ±0.058 b	2.3	0.0	0.072 ± 0.003 a	0.16	0.02
Toxoplasmosis patients (control positive)	30 (15.87%)	32.7 ± 8.45 a	230	5.8	0.10 ± 0.04 b	0.19	0.01
Healthy individuals (control negative)	50 (26.46%)	0.38 ± 0.055 b	2.5	0.0	0.042 ± 0.005 ab	0.13	0.01
LSD value		0.218 *			0.0595 *		
P-value		0.0392			0.0478		
The differences between the various letters in the same column are statistically significant Significant * (P≤0.05), Highly significant ** (P≤0.01).							
Reference range of Toxo IgM: Primary (acute) infection ⩾ 0.6. **Reference range of Toxo IgG**: Secondary (chronic) infection ⩾ 3.0							

Table 2: Specific anti-Toxoplasma IgG and IgM in the case groups.

Groups	Total No. of samples for each group	Mean ± SE pg/ml	Upper Value	Lower Value
Diabetic patients with toxoplasmosis	30	102.3 ± 7.05 c	191.58	25.723
Diabetic patients	30	138.38 ± 5.69 b	185.3	100.38
Toxoplasmosis patients (control positive)	30	115.45 ± 4.44 bc	149.62	88.66
Healthy individuals (control negative)	30	320.43 ± 17.64 a	488.74	222.84
LSD value	32.382 **			
P-value	0.0001			
The differences between the various letters in the same column are statistically significant Highly significant ** (P≤0.01).				

Table 3: Comparison of IL-10 (pg/ml) in the sera of the diabetic patients and control groups.

Discussion

Patients with diabetes are more susceptible to parasitic infections, potentially due to immune system suppression, neuropathy, and poor circulation [23]. Several of the immunological disorders of diabetes have been highlighted in studies of patients with diabetes, including dysregulation of innate immunity, decreased T-cell responses, decreased neutrophil function, and humoral disorders [24]. Therefore, these patients may be susceptible to opportunistic infections such as *T. gondii* infection. Therefore, the timely detection of *T. gondii* in at-risk individuals can help prevent progressive disease and its consequences [25].

The findings for HbA1c in this study are consistent with those of Elkholy *et al.*, [26]. A glycated hemoglobin test can be used for early diagnosis to avoid inexpensive blood tests [27]. HbAlc test is considered an effective method for monitoring diabetes management [28]. Glycated hemoglobin is a routinely utilized marker for long-term glycemic control [29].

Immunocompromised hosts, especially those with weak cell-mediated immunity are at risk of developing widespread disease as a result of reactivation and exacerbation of chronic infections, such as those occasionally seen in diabetic patients [30].

However, in some comparisons there were clear differences when comparing a group of diabetic patients with toxoplasmosis with a control group. In general, interleukins including IL-10, play important roles in the response to injury and infection [31]. Interleukin 10 is an immunomodulatory cytokine that plays a central role in the progression of inflammation. A key role of IL-10 is to act as an essential inhibitory cytokine against the actions of pro-inflammatory cytokines such as IL-12 [32,33].

Interleukin type IL-10 has the ability to prevent the synthesis of pro-inflammatory cytokines such as IL-2, IL-3, IFN-γ, TNF-α and GM-CSF. However, it exhibits a potent capability to suppress the capacity of antigen presenting cells (APCs) and mast cells, thereby inducing maturation of B cells and producing antibodies that also stimulate specific T cells (Th2). Thus, IL-10 produced by mast cells that responds to inflammatory effects [34].

Interleukins and diabetes are closely related to their onset and progression. Variations in the concentration of specific interleukins in the body can indirectly affect the immunological vitality of diabetic patients, which not only aids in the diagnosis, prognosis and treatment of diabetes but also contributes to the development of diabetes. It is also useful for monitoring [35]. Increased T2DM is associated with the secretion of proinflammatory cytokines, and decreased IL-10 gene expression leads to increased production of these cytokines, which is detrimental [36]. The immune response was dominated by IL-10, which suppressed IL-6 and TNF-α production and promoted Th2 cell responses and antibody formation by B lymphocytes [37].

Th2 lymphocytes have specific cytokines (IL-4, IL-5, IL-6, IL-10, IL-13, and IL-13) that can downregulate cell-mediated immune effector mechanisms that are particularly important for host defense against intracellular pathogens especially parasitic disease [38, 39]. Kanash and Yousif [40] studied IL-10 levels in women with breast cancer infected with toxoplasmosis and found that IL-10 levels raised in a group of women with breast cancer and toxoplasmosis, followed by those in women with breast cancer only in comparison with healthy control group which refers to the fact that the presence of *T. gondii* had a profound effect on cellular immune responses through increased IL-10 levels. This is due to the parasite's ability to increase the production of cytokines of Th2 cells including IL-10.

Author contributions

Sarah Ali Saeed: Conceptualization, writing-original draft, methodology, data curation.
Israa Kasim Al-Aubaidi: Supervision, writing-review and editing.

Competing Interests

The authors declared that there were no conflictsof interest.

References

1. Dubey JP. Toxoplasmosis a waterborne zoonosis. Veterinary Parasitology, (2004); 126(1): 57–72.
2. AL-Mossawei MT, AL-Mossawei HM and AL-Dugaily KhY. Serological study of toxoplasmosis spread among unmarried female university using LAT, ELISA and IgG avidity. Baghdad Science Journal, (2016); 13(4): 714–720.
3. Montoya JG and Liesenfeld O. Toxoplasmosis. Lancet, (2004); 363(1): 1965–1976.
4. Al-Maamuri SD, Al-Shanawi FA and Melconian AK. Seroprevalence of Toxoplasma gondii in schizophrenic patients in Iraq using ELISA test. Iraqi Journal Science, (2014); 55(3B): 1243–1248.
5. Saheb EJ, Al-Issa YAH, Mussa IS and Zghair KhH. Incidence of toxoplasmosis in psoriasis patients and possible correlation with tumor necrosis factor – α. Baghdad Science Journal, (2020); 17(1): 214–219.
6. Hade BF, Ghareeb AM and Kawan MH. Direct amplification of B1 gene of Toxoplasma gondii DNA using nested polymerase chain reaction following microwave treatment for whole blood samples. Iraqi Journal Veterinary Medicine, (2015); 39(1): 23–27.
7. Chang Y, Huang Ch and Shiau M. Cytokines, metabolism and type 2 diabetes mellitus. Journal of Biomedical Laboratory Science, (2009); 21(4): 112 – 118.
8. Al-Tukmagi HF and Moussa MA. Quality of life of patients with type II diabetes mellitus in Al-Hilla city-Iraq. Iraqi Journal Pharmaceutical Sciences, (2014); 23(2): 99–103.
9. Fallowfield L. The quality of life: The missing measurement in health care, British Journal General Practice, (1990); 311 pp.

10. Polonsky KS. Lilly Lecture 1994. The beta cell in diabetes: from molecular genetics to clinical research. Diabetes, (1995); 44(6): 705–717.
11. Hanis CL, Boerwinkle E, Chakraborty R, Ellsworth DL, Concannon P, Stirling B, Morrison VA, Wapelhorst B, Spielman RS, Gogolin-Ewens KJ, Shepard JM, Williams SR, Risch N, Hinds D, Iwasaki N, Ogata M, Omori Y, Petzold C, Rietzch H, Schröder HE, Schulze J, Cox NJ, Menzel S, Boriraj VV, Chen X, Lim LR, Lindner T, Mereu LE, Wang YQ, Xiang K, Yamagata K, Yang Y and Bell GI. A genome-wide search for human non-insulin-dependent (type 2) diabetes genes reveals a major susceptibility locus on chromosome 2. Nature Genetics, (1996); 13(2):161–166.
12. Waree P, Ferguson DJ, Pongponratn E, Chaisri U and Sukthana Y. Immunohistochemical study of acute and chronic toxoplasmosis in experimentally infected mice. Southeast Asian Journal Tropical Medicine, (2007); 38(2): 223–231.
13. Waree P. Toxoplasmosis: pathogenesis and immune response. Thailand. Medical Journal, (2008); 8(4): 487–496.
14. Petrak M and Carpenter J. Feline toxoplasmosis. Journal of the American Veterinary Medical Association, (1965); 146: 728–734.
15. Pickup JC. Inflammation and activated innate immunity in the pathogenesis of type 2 diabetes. Diabetes Care, (2004); 27(1): 813–823.
16. Jagannathan M, McDonnell M, Liang Y, Hasturk H, Hetzel J, Rubin D, Kantarci A, Van Dyke TE, Ganley-Leal LM and Nikolajczyk BS. Toll-like receptors regulate B cell cytokine production in patients with diabetes. Diabetologia, (2010); 53(7):1461–1471.
17. Donath MY and Shoelson SE. Type 2 diabetes as an inflammatory disease. Nature Reviews Immunology, (2011); 11(2): 98–107.
18. Miller CM, Boulter NR, Ikin RJ and Smith NC. The Immunobiology of the innate response to Toxoplasma gondii. International Journal for Parasitology, (2009); 39(1): 23–29.
19. Shaw MH, Freeman GJ, Scott MF, Fox BA, Bzik DJ, Belkaid Y and Yap GS. Tyk2 negatively regulates adaptive Th1 immunity by mediating IL-10 signaling and promoting IFN-c dependent IL-10 reactivation. Journal of Immunology, (2006); 176(1): 7263–7271.
20. Khan IA, Matsuura T and Kasper LH. IL-10 mediates immunosuppression following primary infection with Toxoplasma gondii in mice. Parasite Immunology, (1995); 17(4): 185–195.
21. Neyer LE, Grunig G, Remington JS, Rennick D and Hunter CA. Role of interleukin-10 in regulation of T-cell-dependent and T-cell-independent mechanisms of resistance to Toxoplasma gondii. Infection and Immunity (1997); 65(5): 1675–1682.
22. SAS. Statistical Analysis System, User's Guide. Statistical. Version 9.6th ed. SAS. (2018).
23. Akinbo FO, Omoregie R, Egbe E. Intestinal parasitic infections among diabetes mellitus patients. Biomarkers and Genomic Medicine, (2013); 5(1–2):44–7.
24. Saki J, Shafieenia S, Foroutan-Rad M. Seroprevalence of toxoplasmosis in diabetic pregnant women in southwestern of Iran. Journal of Parasitic Diseases, (2016); 40(4): 1586–1589.
25. Saki J, Khodkar I, Shadnoosh F, Safi M, Ghadiri A and Shafieenia Sh. Study of toxoplasmosis in type 2 diabetic patients using ELISA and B1 nested-PCR methods. Annals of parasitology, (2022); 68(2): 367–373.
26. Elkholy AA, Omar RE, Elbadayw AM, Elawady MA and Abou-Ouf E. Investigating the potential link between seroprevalence of Toxoplasma IgG and both types of diabetes mellitus in Benha city, Egypt. Parasitology University Journal, (2022); 15(2): 195–201.
27. Hammed IK, Abed BA and Rashid NF. Glycated hemoglobin as a dual biomarker association between HbA1C and dyslipidemia in type 2 diabetic patients. Journal of Faculty Medicine Baghdad, (2012); 54(1): 88–92.
28. Taher MA, Mosutafa MM and Mahmood AS. Measurements of HbA1c for patients with diabetes mellitus and foot ulceration. Iraqi Journal Pharmaceutical Sciences, (2011); 20(1): 19–24.
29. Alnidawi Bf. Association between glycemic control and serum lipid profile in type 2 diabetic patients: Glycated haemoglobin as a dual biomarker. AL-Kindy College Medicine Journal, (2015); 11(2): 10-14.
30. Cavallazzi LO. Toxoplasmosis of the central nervous system in a diabetic patient. Arq Neuropsiquiatr, (1985); 43(3): 319–321.
31. Mousawy KhM. Possible role of interleukins 6 and 10 in colorectal carcinoma in Iraqi patients. Journal of Faculty Medicine Baghdad, (2004); 47(1): 86–88.
32. Minshawi F, Lanvermann S, MecKenzi E, Jeffery R, Couper K, Papoutsopoulou S, Roers A and Muller W. The generation of an engineered interleukin-10 protein with improved stability and biological function. Frontiers Immunology, (2020); 11(1794): 1–18.
33. Mohamed KI, khadhum MS, Abu-Al-Ess HQ, Mohammed Ali, SH, Al-Fukhar SA, Al-Wattar, W.M.; Hamoudi, S.R. and Mousa, J.M. The effect of Toxoplasma gondii on interleukin-8, interleukin-10, leukotriene B4 and calcium levels in aborted women. International Journal of medical Research and Health Sciences, (2017); 6(11): 76–82.
34. Marietta EV, Chen Y and Weis JH. Modulation of expression of the anti-inflammatory cytokines interleukin-13 and interleukin-10 by interleukin-3. European Journal Immunology, (1996); 26(1): 49–56.
35. Grossmann V, Schmitt VH, Zeller T, Panova-Noeva M, Schulz A, Laubert-Reh D, Juenger C, Schnabel RB, Abt TG, Laskowski R, Wiltink J, Schulz E, Blankenberg S, Lackner KJ, Münzel T and Wild PS. Profile of the immune and inflammatory response in individuals with prediabetes and type 2 diabetes. Diabetes Care, (2015); 38(7): 1356–1364.
36. Naz S, Shafique N, Sharif S, Manzoor F, Saifi S, Firasat S and Kaul H. Association of interleukin 10 (IL-10) gene with type 2 diabetes mellitus by single nucleotide polymorphism of its promotor region G/A 1082. Critical ReviewsTM Eukaryotic Gene Expression, (2020); 30(4): 285–289.
37. Khan IA, Tadashi M and Lioyd HK. IL-10 mediates immunosuppression following primary infection with Toxoplasma gondii in mice. Parasite Immunology, (1995); 7(4): 185–195.
38. Lang C, Gross U and Lüder CG. Subversion of innate and adaptive immune responses by Toxoplasma gondii. Parasitology Research, (2007); 100(2):191–203.
39. Alani MM. Serum interleukin 1 and interleukin 10 levels I Iraqi leukemic patients with hepatitis G virus infection. Journal of Faculty Medicine Baghdad, (2012); 54(4). 340–343.
40. Kanash AK and Yousif JJ. The association of toxoplasmosis and the levels of IL-10 and IL-12 in women with breast cancer. International Journal of Drug Delivery Technology, (2021); 11(2): 265-268.