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Evaluation of Some Cytokines in Patients Have *Toxoplama Gondii* Association with Hepatitis C Virus Infections

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ABSTRACT

Five ml of venous blood samples were collected from a group of 100 individuals diagnosed with Hepatitis C virus (HCV) infection, along with a control group comprising 50 healthy individuals. The sample collection was conducted from Ibn Al-Balady Children & Maternity Hospital. This process spanned from May 2024 to the end of September 2024. The results showed, in the context of our study on Hepatitis C Virus (HCV) and its potential co-infection with Toxoplasmosis, This cohort comprised 100 patients diagnosed with HCV, including 20 who were also co-infected with Toxoplasmosis. The gender and age distribution within this group provided insightful data for our analysis, with 52 males and 48 females ranging from 5 to 65 years. Additionally, for comparative purposes, we included a control group of 50 healthy individuals, balanced in gender (25 males and 25 females) and ranging in age from 5 to 50 years. The demographic data of these participants is crucial for understanding the impact and prevalence of these infections across different segments of the population. The distribution of IL-32, IL-33, and TNF-α among the study groups is thoroughly detailed in Table 4-4 and Figure 4-3. A striking difference is observed in the levels of these cytokines between the control group and the patient group. For IL-32, the control group's mean level was 3.919 with a SE of 0.226. This significantly contrasts with the patient group, which exhibited a much higher mean of 10.428 and a SE of 0.242. The difference in IL-32 levels between the two groups was highly significant, marked by a p-value less than 0.001. Similarly, IL-33 levels showed a substantial disparity. The control group had a mean of 4.775 and a SE of 0.249, whereas the patient group had a notably higher mean of 24.213 and a SE of 0.839. This difference was also highly significant, with a p-value less than 0.001. Lastly, the TNF-α levels followed a similar pattern. The mean level in the control group was 4.655 with a SE of 0.271, compared to the patient group, which had a significantly higher mean of 14.588 and a SE of 0.849. This difference, like the others, was highly significant, indicated by a p-value of less than 0.001.

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1- INTRODUCTION

Toxoplasmosis which is triggered by the parasite Toxoplasma gondii, can be contracted by humans by consuming contaminated food or water, or by coming into contact with feces from infected animals [1]. Although many infections may not show symptoms, toxoplasmosis can result in severe illness in individuals with weakened immune systems, pregnant women, and those with pre-existing liver disease [2]. The exact ways in which T. gondii infection affects the immune response are not fully understood; nevertheless, there is evidence indicating that the parasite can influence the production of cytokines and chemokines, leading to alterations in immune responses [3].

The Hepatitis C virus (HCV) is a virus transmitted through blood that mainly impacts the liver, causing inflammation and possibly resulting in long-term liver issues, cirrhosis, and liver cancer. HCV, an RNA virus, is accountable for liver inflammation and the emergence of hepatocellular carcinoma (HCC). There are six primary genotypes of HCV, with four being prevalent in low-income nations, while genotype 1 is most widespread in middle- and high-income countries [4]. As per information provided by the World Health Organization, over 3% of the global population carries the virus, with over 170 million individuals having chronic infections. The statistics indicate that approximately 85% of individuals progress to chronic hepatitis following acute HCV infection [5]. Contributing to 60-80% of HCV cases in developed countries, the transmission of the bloodborne virus HCV is primarily linked to the frequent usage of intravenous drug [6]. Cytokines are immunomodulatory proteins or glycoproteins produced by a diverse array of cell types. This includes immune cells like macrophages, T or B lymphocytes, and mast cells, as well as non-immune cells such as endothelial cells, fibroblasts, and stromal cells. These cytokines play a pivotal role in the body's response to inflammation and infection [7]. Furthermore, the balance between pro-inflammatory and anti-inflammatory cytokines is crucial. An imbalance in this delicate system can lead to the development and affect the clinical outcomes of a variety of diseases, including infectious diseases, autoimmune disorders, and cancers [8]. The IL-33, IL-32, and TNF genes are responsible for encoding cytokines that are integral in regulating the immune system and mediating inflammation [9].

2- MATERIAL AND METHOD

Five ml of venous blood samples were collected from a group of 100 individuals diagnosed with Hepatitis C virus (HCV) infection, along with a control group comprising 50 healthy individuals. The sample collection was conducted from Ibn Al-Balady Children & Maternity Hospital. This process spanned from May 2024 to the end of September 2024. The study utilized the Enzyme-Linked Immunosorbent Assay (ELISA) technique for measuring HCV IgM, Toxo IgM and IgG, as well as IL-32, IL-33, TNF-α levels. Toxoplasma IgM, IgG antibodies were tested by Elisa technique. The test utilizes recombinant Toxplasma gondii and HCV antigens to selectively detect IgM antibodies to Toxoplasma in serum or plasma as well as IL-32, IL-33, TNF-α levels.

Statistical analysis: The statistical analysis was carried out using the analytical program Graph Pad Prism, and comparisons were done using t test and Qi squire as needed. If the P value was less than 0.05, the data difference was deemed significant, and if it was more than 0.05, the data were deemed non-significant.

3- RESULTS AND DISCUSSION

In the context of our study on Hepatitis C Virus (HCV) and its potential co-infection with Toxoplasmosis, we meticulously selected a cohort of participants to analyse as shown in Table 1. The table belo, titled 'Participant Demographics in HCV and Toxoplasmosis Study,' presents a detailed breakdown of our study group. This cohort comprised 100 patients diagnosed with HCV, including 20 who were also co-infected with Toxoplasmosis. The gender and age distribution within this group provided insightful data for our analysis, with 52 males and 48 females ranging from 5 to 65 years. Additionally, for comparative purposes, we included a control group of 50 healthy individuals, balanced in gender (25 males and 25 females) and ranging in age from 5 to 50 years. The demographic data of these participants is crucial for understanding the impact and prevalence of these infections across different segments of the population.

Table (1: Participant Demographics in HCV and Toxoplasmosis Study

Group	Total Number	HCV Only	Associations HCV & Toxoplasmosis	Gender Distribution	Age Range
Patients with HCV	100	80	20	Males: 52, Females: 48	5-65 years
Healthy Control Individuals	50	-	-	Males: 25, Females: 25	5-50 years

Toxoplasmosis co-infection with Hepatitis C Virus (HCV) refers to a condition where an individual is simultaneously infected with both Toxoplasma gondii, the parasite that causes toxoplasmosis, and the Hepatitis C Virus. Toxoplasmosis is a parasitic disease that typically enters the body through ingestion of undercooked, contaminated meat, exposure from infected cat faeces, or mother-to-child transmission during pregnancy. HCV is a viral infection that primarily affects the liver and is transmitted through exposure to infected blood, such as through injection drug use, blood transfusions, or sexual contact. There is insufficient information regarding the connection between Toxoplasma infection and HCV infection in individuals with chronic liver disease. T. gondii was observed to be more common in individuals with cirrhosis compared to healthy blood donors. Given that blood transfusion can serve as a possible means of transmitting Toxoplasma infection, patients with liver cirrhosis are likely to constitute a group at risk for Toxoplasma infection [10].

Table 2 present the comparative analysis of Toxoplasma IgM and IgG levels among the control and patient groups. For Toxoplasma IgM, the control group had a mean level of 0.111 with a standard error (SE) of 0.024. This was significantly lower compared to the patient group, which had a mean of 0.544 and a SE of 0.187, with a p-value less than 0.05.

Similarly, the Toxoplasma IgG levels showed a marked difference between the two groups. The control group recorded a mean of 0.096 with a SE of 0.022, while the patient group had a notably higher mean of 0.339 and a SE of 0.070. This difference was found to be highly significant, with a p-value of less than 0.001, as also depicted in the same.

Table (2): Distribution of Toxoplasma IgM and IgG among the study groups

Parameter	Groups	No.	Mean ± SE	P value
TOXO IgM	**Control**	50	0.111±0.024	P=0.023 (S)
	HCV Patients	100	0.544±0.187	
TOXO IgG	**HCV Control**	50	0.096±0.022	P= 0.001(HS)
	Patients	100	0.339±0.070	

The distribution of Toxoplasma IgM and IgG in our study aligns with findings from other research, highlighting the varying prevalence and immune response to Toxoplasma infection. For instance, (Sioutas, G. eta al., 2024), [11] analyzed 303 patients, revealing that 71 were positive for IgG and 10 for IgM, mirroring our observations (Nwachukwu,E. et al.,2023), [12] also found similar trends, with 26.7% of seropositive cases having Toxoplasma IgG antibodies and 5.7% with IgM antibodies.

In line with our results, Mousavi-Hasanzadeh et al. (2020) reported that 33.5% of patients in Iraq had IgG Anti-Toxoplasma antibodies. Similarly, [13] noted that 17% of patients in Wasit Province, Iraq, tested positive for ELISA IgG antibodies. These findings underscore the varied prevalence of Toxoplasma antibodies across different regions and populations.

According to Teimouri, A. et al., (2020), [14] the detection of T. gondii-specific IgM is indicative of a recent or current/acute infection, while the presence of T. gondii-specific IgG suggests a past or latent infection. Who further explain that the concurrent presence of both IgG and IgM antibodies typically indicates a recently acquired infection, as IgM antibodies decline soon after the illness is contracted [14].

The observed differences in toxoplasmosis seroprevalence across studies can be attributed to various factors, including geographical location, patient demographics such as age and education level, and lifestyle factors like cat handling, hygiene, and dietary habits, as noted by Cédric et al. (2022) [15]. These factors significantly influence the risk of exposure and subsequent immune response to Toxoplasma gondii.

The distribution of IL-32, IL-33, and TNF-α among the study groups is thoroughly detailed in Table 4-4 and Figure 4-3. A striking difference is observed in the levels of these cytokines between the control group and the patient group. For IL-32, the control group's mean level was 3.919 with a SE of 0.226. This significantly contrasts with the patient group, which exhibited a much higher mean of 10.428 and a SE of 0.242. The difference in IL-32 levels between the two groups was highly significant, marked by a p-value less than 0.001. Similarly, IL-33 levels showed a substantial disparity. The control group had a mean of 4.775 and a SE of 0.249, whereas the patient group had a notably higher mean of 24.213 and a SE of 0.839. This difference was also highly significant, with a p-value less than 0.001. Lastly, the TNF-α levels followed a similar pattern. The mean level in the control group was 4.655 with a SE of 0.271, compared to the patient group, which had a significantly higher mean of 14.588 and a SE of 0.849. This difference, like the others, was highly significant, indicated by a p-value of less than 0.001. These findings are vividly presented in the mentioned table 3 and figure1.

Table (3): Distribution of IL-32, IL-33 and TNF-α among the study groups (patient and controls)

Parameter	Groups	No.	Mean ± SE	P value
IL-32	**Control**	50	3.919±0.226	p<0.0001 (HS)
	Patients	100	10.428±0.242	
IL-33	**Control**	50	4.775±0.249	p<0.0001 (HS)
	Patients	100	24.213±0.839	
TNF-α	**Control**	50	4.655±0.271	p<0.0001 (HS)
	Patients	100	14.588±0.849	

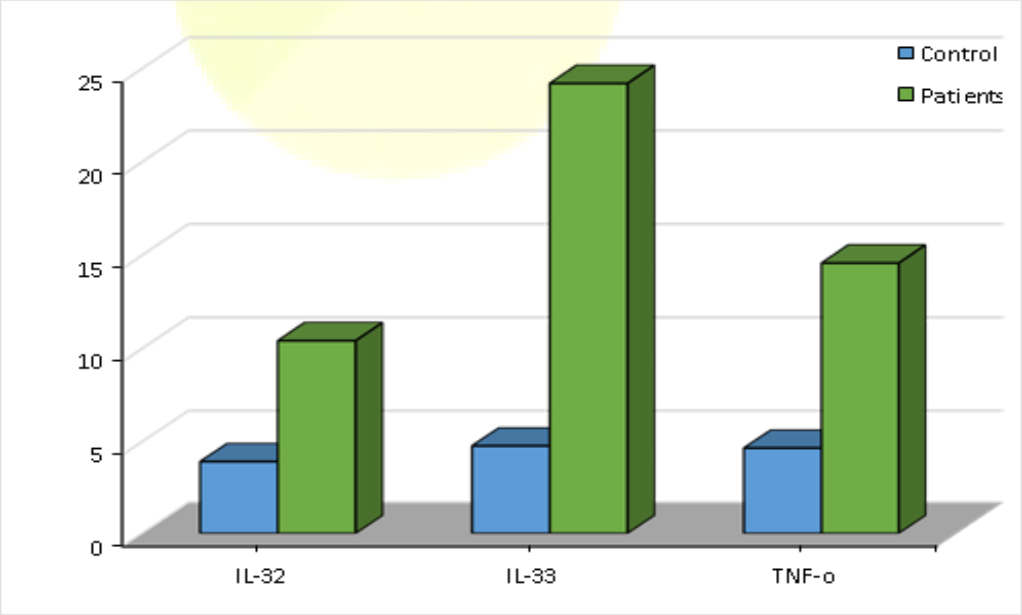

Figure (1): Distribution of IL-32, IL-33 and TNF-α among the study groups (patients and controls)

Cytokines are crucial in regulating immune responses, and their role is particularly significant in the context of hepatitis C virus (HCV) infection. Abnormal levels of cytokines are not only implicated in the progression of HCV but also influence viral persistence and the response to therapy. This is partly due to polymorphisms in cytokine genes, particularly those located within coding or regulatory regions, which can affect cytokine expression and secretion. The impact of these polymorphisms on cytokine levels. Moreover, genes both within and outside the major histocompatibility complex (MHC) are known to affect the nature and magnitude of the immune response. These genes can play a crucial role in the clearance of the hepatitis C virus, as suggested Ferreir, J. et al., (2023), [16].

Focusing on IL-32, a specific cytokine, there are six splice variants: IL-32α, IL-32β, IL-32γ, IL-32δ, IL-32ε, and IL-32ζ, with IL-32α being the most abundant, as noted by IL-32, particularly IL-32α, has been associated with chronic HCV (Shim, S. et al., 2022), [17]. Additionally, Collister et al. (2019) found that peripheral IL-32α levels correlate with hepatic fibrosis scores in these patients implicated IL-32 in the progression of chronic HCV infection [18]. This underscores the broad impact of cytokines like IL-32 in viral infections and immune response. This agrees with other studies by Moschen et al, wherein hepatic IL-32 mRNA expression significantly correlated with hepatic inflammation as determined by serum ALT levels in patients with chronic HCV [19].

The studies on interleukin-33 (IL-33) and tumour necrosis factor-alpha (TNF-α) offer valuable insights into their roles in hepatitis C virus (HCV) infection and associated liver damage. These findings underscore the complex interplay of cytokines like IL-33 and TNF-α in the immune response and pathogenesis of HCV infection and liver damage. Understanding these dynamics is crucial for developing targeted therapies and managing HCV and its complications effectively (Hassoon, H. J. 2024), [20].

Table (4) provides an interesting insight into the distribution of anti-Toxoplasma IgM and IgG antibodies across different age groups in both control subjects and toxoplasmosis patients. In the control group, the mean IgM levels across the age groups of 5-20, 21-35, and 36-50 years did not show significant differences, indicating a relatively stable exposure or immune response to Toxoplasma in the general population across these ages. Similarly, in toxoplasmosis patients, the mean IgM levels did not vary significantly across all age groups, including 5-20, 21-35, 36-50, and 50-65 years. However, the highest prevalence was observed in the 5-20 years age group.

Table (4): Distribution of anti-Toxoplasma IgM and IgG among the study groups according to age

Parameter		No.	Age groups	Mean + SE	P value
TOXO IgM	Control	50	5-20	0.088±0.030	0.79 (NS)
			21-35	0.054±0.023	
			36-50	0.200±0.062	
	Patients	100	5-20	0.677±0.547	0.89 (NS)
			21-35	0.612±0.391	
			36-50	0.250±0.094	
			50-65	0.575±0.243	
TOXO IgG	Control	50	5-20	0.065±0.019	0.71 (NS)
			21-35	0.070±0.054	
			36-50	0.171±0.051	
	Patients	100	5-20	0.180±0.065	0.28 (NS)
			21-35	0.374±0.134	
			36-50	0.566±0.217	
			50-65	0.236±0.094	

NS: Non-significant

Our study found the highest seroprevalence of Toxoplasma IgM in the 5-20 years age group and the highest Toxoplasma IgG seroprevalence in the 36-50 years age group, the differences among various age groups were not statistically significant. Similarly, a study by Ivănescu et al. (2021) highlighted that the peak prevalence of anti-T. gondii IgM antibodies was in those under 20 years (5.26%) during 2013-2016, shifting to the above 35 years age group (1.76%) in 2019-2022 [21].

However, Uchôa et al. (2022) reported a contrasting trend, with a decline in prevalence after 30 years, and a higher rate noted between 20 and 29 years. Despite these variations, the majority of studies, including a meta-analysis concur that T. gondii IgG seroprevalence tends to rise with age, possibly due to increased cumulative exposure over time [22].

Toxoplasmosis infection rates generally escalate with advancing age, but there is considerable geographical variation in these rates, influenced by factors like local health standards, dietary practices, and socioeconomic conditions. Some regions have witnessed a decline in toxoplasmosis prevalence, attributed to improvements in hygiene and agricultural practices, as noted by Yang, Z. et al., (2024)**,** [23].

IgM antibodies are typically indicative of recent or acute infection, suggesting that younger individuals in this cohort may have been more recently infected. When comparing our study's findings on the age-related distribution of anti-Toxoplasma IgM and IgG antibodies with those reported by Colzato et al. (2021), interesting contrasts and similarities emerge. Colzato et al. highlight that approximately one-third of the global population has latent toxoplasmosis, which is more prevalent in older individuals due to its lifelong persistence. They suggest that while most infected individuals are asymptomatic, T. gondii might induce cognitive changes across the lifespan. In our study, the prevalence of anti-Toxoplasma IgM, indicative of recent or acute infection, was highest in the younger age group (5-20 years) among toxoplasmosis patients. This contrasts with Colzato et al.'s emphasis on the prevalence in older age due to chronic, lifelong infection. Regarding anti-Toxoplasma IgG antibodies, in the control group, the mean IgG levels across the age groups of 5-20, 21-35, and 36-50 years were relatively low and showed no significant differences [24]. This suggests a low level of past exposure to Toxoplasma across these ages in the general population. In toxoplasmosis patients, while there were no significant differences in mean IgG levels across the age groups, the 36-50 years age group showed the highest prevalence Mustafa, K. M.e et al., (2024), [25]. IgG antibodies indicate past exposure and possibly chronic infection, which could imply that individuals in this age group have been exposed to Toxoplasma for a longer duration or had earlier infections that have persisted. These results enhance understanding of Toxoplasma gondii seroprevalence across ages, suggesting targeted healthcare strategies based on higher IgM in younger and IgG in middle-aged toxoplasmosis patients [25].

4- CONCLUSION

This comprehensive study has provided valuable insights into the complex interplay of cytokines, oxidative stress markers, and genetic polymorphisms in the context of Hepatitis C (HCV) and potential co-infection with Toxoplasmosis. Key findings indicate that the expression levels of IL33, IL32, and TNF genes are significantly altered in HCV patients, especially those co-infected with Toxoplasmosis.

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تقييم بعض السيتوكينات في المرضى المصابين بطفيلي التوكسوبلازما جوندي المرتبط بعدوى فيروس التهاب الكبد الوبائي سي

الخلاصة:

تم جمع خمسة ملم مكعب من عينات الدم الوريدي من مجموعة مكونة من 100 فرد تم تشخيص إصابتهم بعدوى فيروس التهاب الكبد الوبائي سي (HCV)، إلى جانب مجموعة تحكم تضم 50 فردًا سليمًا. تم جمع العينة من مستشفى ابن البلدي للأطفال والولادة. امتدت هذه العملية من مايو 2024 إلى نهاية سبتمبر 2024. أظهرت النتائج، في سياق دراستنا حول فيروس التهاب الكبد الوبائي سي (HCV) والعدوى المحتملة المشتركة مع داء المقوسات. ضمت هذه المجموعة 100 مريض تم تشخيص إصابتهم بفيروس التهاب الكبد الوبائي سي، بما في ذلك 20 مصابًا أيضًا بداء المقوسات. وقد قدم توزيع الجنس والعمر داخل هذه المجموعة بيانات مفيدة لتحليلنا، حيث كان هناك 52 ذكرًا و48 أنثى تتراوح أعمارهم بين 5 إلى 65 عامًا. بالإضافة إلى ذلك، ولأغراض المقارنة، قمنا بتضمين مجموعة تحكم مكونة من 50 فردًا سليمًا، متوازنين في الجنس (25 ذكرًا و25 أنثى) وتتراوح أعمارهم بين 5 إلى 50 عامًا. تعد البيانات الديموغرافية لهؤلاء المشاركين أمرًا بالغ الأهمية لفهم تأثير وانتشار هذه العدوى عبر شرائح مختلفة من السكان. تم تفصيل توزيع IL-32 و-IL 33 وTNF-α بين مجموعات الدراسة بدقة في الجدول 4-4 والشكل 4-3. لوحظ فرق ملحوظ في مستويات هذه السيتوكينات بين مجموعة التحكم ومجموعة المرضى. بالنسبة لـ IL-32، كان متوسط مستوى مجموعة التحكم 3.919 مع خطأ معياري قدره 0.226. يتناقض هذا بشكل كبير مع مجموعة المرضى، التي أظهرت متوسطً أعلى بكثير بلغ 10.428 وخطأ معياري قدره 0.242. كان الاختلاف في مستويات IL-32 بين المجموعتين مهمًا للغاية، حيث تميز بقيمة p أقل من 0.001. وبالمثل، أظهرت مستويات IL-33 تباينًا كبيرًا. كان متوسط المجموعة الضابطة 4.775 و0.249 SE، في حين كان متوسط مجموعة المرضى أعلى بشكل ملحوظ 24.213 و0.839 SE. كان هذا الاختلاف أيضًا مهمًا للغاية، حيث كانت القيمة p أقل من 0.001. أخيرًا، اتبعت مستويات TNF-α نمطًا مشابهًا. كان متوسط المستوى في المجموعة الضابطة 4.655 مع SE 0.271، مقارنة بمجموعة المرضى، التي كان متوسطها أعلى بكثير 14.588 و0.849 SE. كان هذا الاختلاف، مثل غيره، مهمًا للغاية، حيث تم الإشارة إليه بقيمة p أقل من 0.001.