Open Access Research Article

Assessment of Interleukin-17 levels in patients with hepatitis C Viral Infection

Doi: https://doi.org/10.32007/jfacmedbagdad.2157

Rafal M. Lafta*[1] Zainab A. Hamid[2]

[1]*Department of Microbiology, College Medicine, University of Baghdad, Baghdad, Iraq.*
[2]*Medical City/ Gastroenterology and Hepatology, Teaching Hospital Baghdad, Iraq*

J Fac Med Baghdad 2024; Vol.66, No. 1
Received: June, 2023
Accepted: Sept., 2023
Published:April .2024

Abstract:

Background: The roll of IL-17 during chronic hepatitis C virus (HCV) infection is not well understood. Elevated levels of IL-17 may increase the risk of liver fibrosis development in chronic HCV infection. However, the exact mechanism of how IL-17 contributes to the progression of HCV infection remains unclear.

Objectives: The aim of this study was to examine the association between HCV infection and IL-17. This was done by measuring the serum levels of interleukins and the viral load of HCV in patients with HCV infections.

Patients and methods: The study included fifty HCV patients who were receiving treatment at the Gastroenterology and Hepatology Teaching Hospital / Medical City Complex in Baghdad. The study was conducted between November 2022 and April 2023, and the study groups were matched with 50 controls based on age and sex. Seven milliliters of venous blood samples were taken from all participants to test for anti-HCV antibodies using ELISA kit, quantitative measurement of HCV RNA by PCR, and estimation of serum levels of IL-17.

Results: The range of HCV viral RNA titers among the patients was 165-55595000 copies/ml, and all of them tested positive for anti-HCV antibodies. On the other hand, the control group tested negative for the virus. The mean concentration of IL-17 in the patients' group was 1458.03(±165.7) pg/ml, while the control group had a mean of 628.04 (±163.0) pg/ml ($P < 0.0001$). The R-value for HCV viral load and IL-17 was 0.1388. It is worth noting that the mean serum IL-17 concentration was highest among the patients aged ≥ 50 years old. As for the HCV patients on treatment, their mean viral load was 406, 3051.90 copies/ml, and their mean IL-17 concentration was 1444.56 pg/ml. The patients who did not receive treatment had a mean higher mean viral load of 765,525.94 copies/ml, and their mean IL-17 concentration was 1484.19 pg/ml.

Conclusion: The concentration of IL-17 was found to be significantly higher in patients with Hepatitis C virus (HCV), and it showed a weak positive correlation with HCV viral load. Moreover, older patients with HCV had higher levels of IL-17 in their blood. However, the measurement of HCV viral load and IL-17 did not show any significant differences in their readings.

Keywords: IL-17; Hepatitis C virus; HCV related interleukins; chronic hepatitis.

Introduction:

Hepatitis C virus (HCV) infection is an infectious disease mainly affecting the liver and may end with liver cirrhosis and Hepatocellular cancer [1]. The main methods of viral transmission include hazardous contaminated injection techniques, dental and surgical procedures, and blood transfusions. The hemodialysis setting has certain characteristics that make it easier to spread HCV, such as a high chance of blood getting on surfaces, equipment, and devices, and a large number of patients receiving treatment at once in a communal area. At Hemodialysis institutions, newly acquired HCV infections (also known as seroconversions) are not unusual [2]. Around 80% of HCV infections result in chronic hepatitis. HCV infection induces chronic inflammation, endothelial invasion and dysfunction, and changes in serum Levels of inflammatory and proinflammatory biomarkers [6].

Both the innate immune and adaptive immune responses contribute to HCV infection and they induce the production of certain cytokines [7]. Many cellular molecules and soluble cytokines showed relationships to HCV infections like for example CTLA-4, PD-1, and PD-L1 immune checkpoint biomarkers as predictors for renal complications [8]; the intercellular adhesion molecule-1, N-terminal pro-brain natriuretic peptide and cardiac troponin-I as predictors for cardiovascular factors [9].

Interleukin -17 (IL-17) is a proinflammatory cytokine that is produced from T-helper type 17 (Th-17) cells. This cytokine plays an important role in

*Correspondent Author:
ZainabA.Hamid@comed.uobaghdad.edu.iq

the regulation of host immune responses against many pathogens particularly against viral infections [10]. Furthermore, dysregulation in the production of IL-17 induces chronic inflammation and autoimmune disorders, and may lead to cirrhosis, fibrosis, and hepatocellular carcinoma (HCC) [11]. Several researches showed that IL-17 is not only involved in the antiviral immune response but also facilitates virus-mediated infections [12, 13].

The functions of IL-17 and IL-35 during chronic hepatitis C virus (HCV) infection are poorly understood. Serum IL-17 levels in individuals with persistent HCV infection were considerably greater than in controls. This finding supports the hypothesis that IL-17 plays a role in the etiology and/or development of liver fibrosis by increasing the level of serum IL-17 in liver damage caused by chronic hepatitis and cirrhosis [14].

The current study aimed at testing the association of IL-17 with HCV infections by measuring serum levels of this interleukin as well as the viral load of HCV in patients with HCV infections.

Methodology:

This study enrolled 100 individuals in two study groups, the HCV patients' group (n=50) and the healthy control group (n=50). The HCV patients were age and sex-matched with the control group.

The HCV patients were attending Gastroenterology and Hepatology Teaching Hospital / Medical City Complex in Baghdad / Iraq. The control group included healthy individuals who accompanied the patients in the hospital. The study extended between November 2022 and April 2023. Some HCV patients were receiving directly acting anti-HCV medications. Patients were identified as having a chronic HCV infection through anti-HCV testing and met the inclusion criteria for the study. The exclusion criterion was the presence of concomitant comorbidities.

The patients were regularly screened for HCV by using ELISA technique, already Anti-HCV positive samples were confirmed by real-time polymerase chain reaction (PCR). A questionnaire was designed to record demographic data from each participant in the study.

Seven milliliters of venous blood sample were collected by medical staff from HCV patients and control individuals infected with HCV. The study investigated anti-HCV antibodies using ELISA kit (Hightop Biotech company/China), quantitative measurement of HCV RNA by PCR (Cepheid A company/ Sweden), and estimation of serum levels of IL-17 (Sunlong Biotech company/China). Seven milliliters of blood were aspirated by venipuncture and then collected into sterile gel tubes, the blood was centrifuged, then the serum was divided into three tubes, 0.5 milliliters of serum in each tube; the first tube for the detection of anti-HCV antibodies, the second tube for quantitative detection of HCV RNA, and the third tube for quantitative measurement of serum IL-17. The sera were immediately frozen at -20°C until used.

The Statistical Package for Social Sciences (SPSS vs 21 for Mac. IBM Inc. Chicago) was used for statistical analyses. The software available online was used to determine the correlation coefficient. Statistical significance was defined as $p<0.05$.

Results:

The HCV patients included 28 males and 22 females, who were age and sex matched with the control group. The HCV patients were between 18-51 years of age. All the subjects in the control group were negative for HCV viral load with zero copies/ml results. All the HCV patients tested positive for HCV viral RNA titers ranging between 165-55595000 copies/ml with a mean and median viral load of 2941893.08 copies/ml and 236045.5 copies/ml respectively, table (1).

Table (1): HCV viral load results among HCV patients

Statistics	HCV viral load
Mean	2941893.08±753819.1
Median	236045.5
Range	55594835 (55595000-165)
Mode	All values appeared just once
Geometric Mean	253076.90260865
Count	50

All the HCV patient tested positive for anti-HCV antibodies while the controls tested negative for these antibodies, table (2).

Table (2): The positivity of anti-HCV antibodies in study groups using ELISA technique

Study group	Positive anti-HCV Antibodies No. (%)	Negative anti-HCV Antibodies No. (%)
Patients group	50 (100%)	0 (0%)
Control group	0 (0%)	50 (100%)

The mean serum concentration of IL-17 in the HCV patients was 1458.03926 (±165.7) ng/ml while for control group it was 628.042825 (±163.0) ng/ml. The difference in the means of serum IL-17 between the patients and controls was statistically significant ($P < 0.0001$). The median and range values of IL-17 was higher in the patient than the controls, table (3).

Table (3): The statistics for serum interleukin-17 concentration in HCV patients and controls

Statistics	HCV patients' group	Control group
Mean (±SD) *	1458.039 (±165.72)	628.043 (±163.04)
Median	1463.928	640.4545
Range	763.032	541.806
Largest	1897.062	954.297
Smallest	1134.03	412.491
Count	50	50

** P value <0.0001*

The correlation coefficient between HCV viral load and IL-17 was 0.1388, which is weak and not statistically significant.

Gender distribution of HCV viral load concentration revealed a mean concentration in males equal to

4103,393.82 (±10740307.46), while for females it was 1463619.41 (±4467404.8). The difference between the two means was not significant (P = 0.2856), table (4).

Table (4): HCV viral load statistics in male and female patients

Statistics	HCV viral load	
	Males (n=28)	Females (n=22)
Mean ±SD	4103,393.821 (±10740307.46)	1463,619.414 (±4467404.82)
Median	1427934	118000
Range	55594770	21399835
Mode	All values appeared just once.	All values appeared just once.
Largest	55595000	21400000
Smallest	230	165

**P = 0.2856*

The largest age group among HCV patients was in those between 30-39 years (23/50), while the least was in those <20 years old (n=1) and ≥50 years (n=1). The frequencies in age groups 20-29 years old and 40-49 years old were n=14 and n=11 respectively. The highest mean HCV viral load (3,373,287 copies/ml) was in age group 40-49 years old, while the least mean HCV viral load (64,949 copies/ml) was in group <20 years old; figure (1).

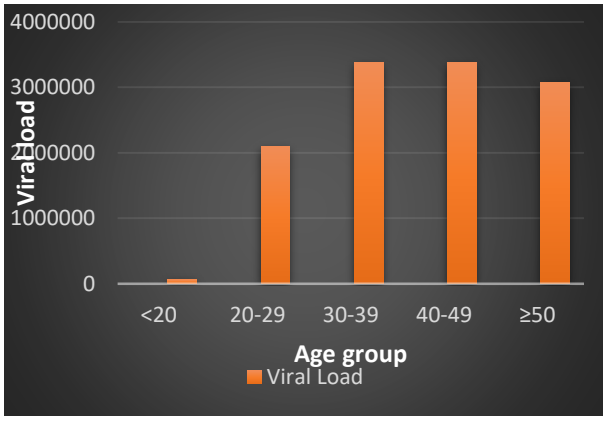

Figure (1): The means of HCV Viral load titers among different age groups

The mean serum IL-17 concentration was in its highest value in age group ≥ 50 years old while the lowest value was in age group <20 years old, figure (2).

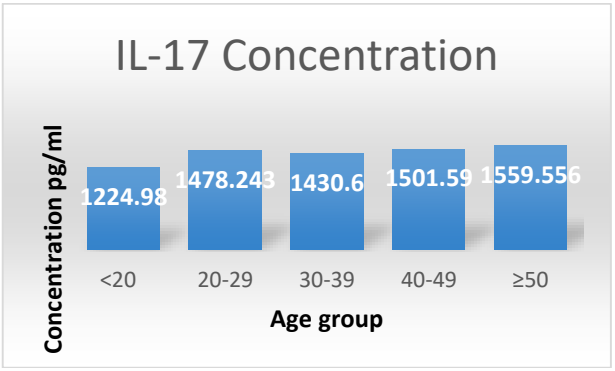

Figure (2): The means of IL-17 concentrations (pg\ml) in different age groups among HCV patient

The correlation coefficient (R) between the HCV patients ages and their viral load was equal to 0.0943, which indicates a weak correlation.

When the IL-17 concentrations for HCV patients were aligned with their ages, the correlation coefficient (R) was 0.2051; while the R value for interleukin 17, although technically positive correlations, the relationship with patients' ages was weak.

The patients were categorized into two subgroups, those on treatment [n=33 (66%)] and those without treatment [n=17 (34%)]. Those on treatment had a mean viral load of 406,3051.90 copies/ml and a mean IL-17 concentration of 1444.56 ng/ml, while those without treatment had a mean viral load of 765,525.94 copies/ml and a mean IL-17 concentration of 1484.19 ng/ml. The differences in the means of the two subgroups were statistically not significant (P values = 0.2661 and 0.4814 respectively). Moreover, the HCV treatment did not make significant changes in means of IL-17 concentrations.

Discussion:

In this study, quantitative PCR method was applied for detection and quantification of HCV viral load; this technique is of higher sensitivity than qualitative PCR methods [15]. The smallest HCV viral load was 165 copies/ml which represent persistent viremia [16], which questions the efficacy of the anti-HCV drugs administered to the patients as 66% of them were receiving anti-HCV medications. It may be the result of recent administration of anti-HCV medications that didn't give enough time (three months) to produce a rapid virologic response. In contrast to this finding, Carver et al reported a high rate of sustained viral response after receiving directly-acting anti-HCV medication [17]. The PCR results revealed high viral load among HCV patients, however, with the new anti-HCV medications a rapid decline in viral load can be achieved within 2-3 weeks from starting treatment and can reach to undetectable level [18].

The results revealed that all the patients with HCV infections have positive anti-HCV antibodies; which is in agreement with other studies indicating that

those who have a chronic HCV infection would have anti-HCV antibodies that last forever [19].

Interleukin-17 connects the recruitment and activation of neutrophils with the activation of T cells; as a result, IL-17 can influence the pathophysiology of inflammatory disorders or mediate the protective innate immunity against pathogens [20]. In the current study, there was a significant elevation in serum IL-17 among HCV patients when compared to the control group. Foster et al had reported low levels of IL-17 among chronically infected HCV patients with these levels close to healthy controls [21]. On the other hand, Askoura et al found an increase in IL-17 in HCV infections with hepatocellular carcinoma (HCC) but not in HCV with liver fibrosis, and they suggested a relation between IL-17 and the development of HCC in HCV patients [22]. These findings suggest a possible role of IL-17 in HCV pathogenesis. Future studies are needed for better elucidation of IL-17 role in HCV infection.

Cytokines play a crucial role in the development of viral infection by taking part in the induction and effector phases of all inflammatory and immunological responses. Antiviral inflammation is heavily influenced by excessive, inadequate, or inappropriate cytokine responses [23].

The results of this study showed a weak positive correlation between IL-17 and HCV viral load, which can be explained by the fact that the higher the HCV viral load, the stronger the immune response and higher IL-17 secretion as an inflammatory mediator. However, as the HCV infection becomes more chronic, IL-17 release starts to decline as other regulatory mediators begin to be released which control the IL-17 release. Gomaa et al recorded a significant correlation between IL-17 and HCV viral load [24].

The non-significant differences between the means of viral load for HCV among males and females suggest that hormonal differences do not appear to influence HCV viral load. However, a systematic review by Abdel-Gawad had shown that males have higher viral load than females [25].

The age of the population may have an impact on the distribution of HCV infection. Age distribution was shown to be correlated with HCV genotypes in several studies; for instance, subtypes 1a and 1b were more prevalent in older patients (51–60 years old), but subtype 3b was the most prevalent subtype in younger people (10 - 20 years old) [26]. Our study's findings demonstrated that the frequency of HCV infection varied with age. The current study did not address HCV genotyping; therefore, we cannot attribute age distribution to HCV genotypes. The age distribution disparities may be related to the timing of HCV screening for the group after surgical intervention or premarital screening, which occurs more often in young individuals.

The progression to cirrhosis is hastened and happens more frequently in patients who acquire the virus at an old age [27].

The current study found that increase in mean HCV viral load with age was also associated with increase in serum IL-17 concentration, which is logical as an increase in viral load will be associated with more inflammatory response and more secretion of inflammatory markers including IL-17. However, the correlations between HCV patients' ages and interleukin concentrations were not significant, which may be due to other factors influencing their levels. To the best of our knowledge, the current study was the first to assess the serum IL-17 concentrations in different age groups of HCV patients.

Sustained virologic response (SVR), the undetectable HCV RNA 12 weeks after the end of antiviral treatment, is the aim of HCV treatment. SVR is assessed by measuring the HCV viral load which is more likely linked to low viral load [28]. Rapid virologic response (RVR), which is defined as undetectable HCV RNA at four weeks of therapy, is a potent tool for predicting treatment success [29]. Unfortunately, none of the HCV patients in the current study reached the RVR during the period of the study. The viral load and cytokines were measured only one time, and the starting time, which may be less than four weeks, was not recorded, thus it may need another measurement to predict the changes in these variables.

Author Contributions

Author Contributions: "Conceptualization, Zainab A. Hamid ,Rafal Mohammed Lafta; methodology Rafal Mohammed Lafta . Validation, Zainab A. Hamid. Formal analysis, Zainab A. Hamid.; investigation, Rafal Mohammed Lafta; resources, Zainab A. Hamid.; data curation, Zainab A. Hamid writing original draft preparation, Rafal Mohammed Lafta and Zainab A. Hamid .writing. Rafal Mohammed Lafta, and Zainab A. Hamid.; visualization, Zainab A. Hamid.; Supervision, Zainab A. Hamid.; project administration, funding acquisition, Rafal Mohammed Lafta, and Zainab A. Hamid.

Funding: no funding

Conflicts of Interest, "no conflict of interest.

Authors' declaration:

We confirm that all the Figures and Tables in the manuscript belong to the current study. Besides, the Figures and images, which do not belong to the current study, have been given permission for re-publication attached to the manuscript. Authors sign on ethical consideration's approval-Ethical Clearance: The project was approved by the local ethical committee of Department of Microbiology/ College of Medicine/ University of Baghdad, according to the code number (0210) on (25/ 06/ 2023).

References:

[1] Maasoumy B, Wedemeyer H. Natural history of acute and chronic hepatitis C. Best Pract Res Clin Gastroenterol. 2012 Aug;26(4):401-12. https://doi.org/10.1016/j.bpg.2012.09.009

[2] Nguyen DB, Bixler D, Patel PR. Transmission of hepatitis C virus in the dialysis setting and strategies for its prevention. Semin Dial. 2019 Mar;32(2):127-134. https://doi.org/10.1111/sdi.12761

[3] Gupta E, Bajpai M, Choudhary A. Hepatitis C virus: Screening, diagnosis, and interpretation of laboratory assays. Asian J Transfus Sci. 2014 Jan;8(1):19-25.https://doi.org/10.4103/0973-6247.126683

[4] Al-Bayaa Y, Hamid Z, Bayati A, Zeiny S. The Incidence of Hepatitis C Virus Infections among People Screened in Governmental Health Care Facilities in 2018 in Iraq. Indian Journal of Forensic Medicine & Toxicology, July-September 2020, Vol. 14, No.3. https://medicopublication.com/index.php/ijfmt/article/view/10565/9902

[5] Rana Akram Hadi, Zainab A. Hamid. The Frequency of Hepatitis C Viral Infections and its Correlation with IL-12 and IL-18 among Major Thalassemic Patients in Baghdad. Egypt. J. Chem. (2022); Vol. 65, No. 8, pp. 593 – 600. DOI:10.21608/EJCHEM.2021.112813.5126

[6] Babiker A, Jeudy J, Kligerman S, Khambaty M, Shah A, Bagchi S. Risk of Cardiovascular Disease Due to Chronic Hepatitis C Infection: A Review. J Clin Transl Hepatol. 2017 Dec 28;5(4):343-362. https://doi.org/10.14218/JCTH.2017.00021

[7] Dustin LB. Innate and Adaptive Immune Responses in Chronic HCV Infection. Current Drug Targets 2017. 18, 826–843. https://doi.org/10.2174/1389450116666150825110532

[8] Jouda RS, Ibrahim B M, Al-Khafagi AF. Role of CTLA-4, PD-1 and PD-L1 immune biomarkers among HCV patients undergoing hemodialysis. Biomedicine. 2023 Mar. 28;43(01):428-32. https://doi.org/10.51248/.v43i01.2112

[9] Salman HS, Ibrahim BM, and Al-Khafagi AF. Serum Levels of Intercellular Adhesion Molecule-1, N-Terminal pro-Brain Natriuretic Peptide and Cardic Troponin-I among Haemodialysis Patients with Hepatitis C Virus. Journal of Cardiovascular Disease Research; 2021,Vol. 12, issue (5): 696-700. https://www.researchgate.net/publication/357033117_Serum_Levels_of_Intercellular_Adhesion_Molecule-1_N-Terminal_pro-rain_Natriuretic_Peptide_and_Cardic_Troponin-I_among_Haemodialysis_Patients_with_Hepatitis_C_Virus

[10] Ge Y, Huang M, Yao YM. Biology of Interleukin-17 and Its Pathophysiological Significance in Sepsis. Front Immunol. 2020 Jul 28;11:1558. https://doi.org/10.3389/fimmu.2020.01558

[11] Beringer A, Miossec P. IL-17 and IL-17-producing cells and liver diseases, with focus on autoimmune liver diseases. Autoimmun Rev. 2018 Dec;17(12):1176-1185. https://doi.org/10.1016/j.autrev.2018.06.008

[12] Lee MH, Chang JT, Liao C, Chen Y, Kuo M, and Shen C. Interleukin 17 and peripheral IL-17-expressing T cells are negatively correlated with the overall survival of head and neck cancer patients. Oncotarget.2018 Feb 9; 9(11): 9825–9837. https://doi.org/10.18632/oncotarget.23934

[13] Ma WT, Yao XT, Peng Q, Chen DK. The protective and pathogenic roles of IL-17 in viral infections: friend or foe? Open Biol. 2019 Jul 26;9(7):190109. https://doi.org/10.1098/rsob.190109

[14] Chang Q, Wang YK, Zhao Q, Wang CZ, Hu YZ, Wu BY. Th17 cells are increased with severity of liver inflammation in patients with chronic hepatitis C. J Gastroenterol Hepatol. 2012;27(2):273–278. https://doi.org/10.1111/j.1440-1746.2011.06782.x

[15] Caliendo AM, Schuurman R, Yen-Lieberman B, Spector SA, Andersen J, Manjiry R, et al. CMV Working Group of the Complications of HIV Disease RAC, AIDS Clinical Trials Group. Comparison of quantitative and qualitative PCR assays for cytomegalovirus DNA in plasma. J Clin Microbiol. 2001 Apr;39(4):1334-8. https://doi.org/10.1128/JCM.39.4.1334-1338.2001

[16] Hahn JA, Tully DC, Evans JL, Morris MD, Briceno A, Bean DJ, et al. Role of HCV Viremia in Corroborated HCV Transmission Events Within Young Adult Injecting Partnerships, Open Forum Infectious Diseases, Volume 6, Issue 4, April 2019, ofz125. https://doi.org/10.1093/ofid/ofz125

[17] Carver AB, Zuckerman AD, DeClercq J, Choi L, Chastain CA. Incidence and Impact of Persistent Viremia on SVR Rates in Patients Receiving Direct-Acting Antiviral Therapy. Open Forum Infect Dis. 2020 Nov 26;7(12):569. https://doi.org/10.1093/ofid/ofaa569

[18] Kaur D, Prabhakar K, and Das S. Distribution of HCV genotypes and HCV RNA viral load in hepatitis infected patients of Kolar region, Karnataka, India. Bioinformation. 2022; 18(4): 387–391. https://doi.org/10.6026/97320630018387

[19] Gupta E, Bajpai M, and Choudhary A. Hepatitis C virus: Screening, diagnosis, and interpretation of laboratory assays. Asian J Transfus Sci. 2014 Jan-Jun; 8(1): 19–25. https://doi.org/10.4103/0973-6247.126683

[20] Zenobia C, Hajishengallis G. Basic biology and role of interleukin-17 in immunity and inflammation. Periodontol 2000. 2015 Oct;69(1):142-59. https://doi.org/10.1111/prd.12083

[21] Foster RG, Golden-Mason L, Rutebemberwa A, Rosen HR. Interleukin (IL)-17/IL-22-Producing T cells Enriched Within the Liver of Patients with Chronic Hepatitis C Viral (HCV) Infection. Dig Dis Sci **57**, 381–389 (2012). https://doi.org/10.1007/s10620-011-1997-z

*[22] Askoura M, Abbas HA, Al Sadoun H, Abdulaal WH, Abu Lila AS, Almansour K, et al. Elevated Levels of IL-33, IL-17 and IL-25 Indicate the Progression from Chronicity to Hepatocellular Carcinoma in Hepatitis C Virus Patients. Pathogens **2022**, 11, 57. https://doi.org/10.3390/pathogens11010057*

[23] Kany S, Vollrath JT, Relja B. Cytokines in Inflammatory Disease. Int J Mol Sci. 2019 Nov 28;20(23):6008. https://doi.org/10.3390/pathogens11010057

*[24] Gomaa AF, Wahba MO, Hafez RA, Eldaly OM, Badran SG. Assessment of the role of interleukin 17A and interleukin 17F in chronic hepatitis C virus infection in Egyptian patients. Egypt J Intern Med **31**, 199–202 (2019). https://doi.org/10.4103/ejim.ejim_119_18*

[24] Abdel-Gawad M, Nour M, El-Raey F, Nagdy H, Almansoury Y, El-Kassas M. Gender differences in prevalence of hepatitis C virus infection in Egypt: a systematic review and meta-analysis. Sci Rep. 2023 Feb 13;13(1):2499. https://doi.org/10.1038/s41598-023-29262-z

[25] Niu Z, Zhang P, Tong Y. Age and gender distribution of Hepatitis C virus prevalence and genotypes of individuals of physical examination in WuHan, Central China. Springerplus. 2016 Sep 13;5(1):1557. https://doi.org/10.1186/s40064-016-3224-z

[26] Cainelli F. Hepatitis C virus infection in the elderly: epidemiology, natural history and management. Drugs Aging. 2008;25(1):9-18. https://doi.org/10.2165/00002512-200825010-00002

*[27] AASLD-IDSA HCV Guidance Panel Hepatitis C Guidance 2018 Update: AASLD-IDSA Recommendations for Testing, Managing, and Treating Hepatitis C Virus Infection. Clin. Infect. Dis. 2018;**67**:1477–1492. doi: 10.1002/hep.31060*

*[28] European Association for the Study of the Liver EASL recommendations on treatment of hepatitis C: Final update of the series. J. Hepatol. 2020;**73**:1170–1218. https://doi.org/10.1016/j.jhep.2020.08.018*

How to Cite this Article
Assessment of Interleukin-17 levels in patients with hepatitis C Viral Infection. JFacMedBagdad [Internet]. [cited 2024 Mar. 19];66(1). Available from: https://iqjmc.uobaghdad.edu.iq/index.php/19JFacMedBaghdad36/article/view/2157

تقييم مستويات انترلوكين ١٧ في مرضى التهاب الكبد الفايروسي نوع سي

رفل محمود محمد / م. بغداد التعليمي
أ.م.د. زينب عبد الجسين / كلية الطب / جامعة بغداد/ قسم الاجياء المجهرية

خلاصة:

الخلفية: إن دور IL-17 أثناء الإصابة بفيروس التهاب الكبد الوبائي المزمن (HCV) ليس مفهوما جيدا. قد تزيد المستويات المرتفعة من IL-17 من خطر الإصابة بتليف الكبد في عدوى فيروس التهاب الكبد الوبائي المزمن. ومع ذلك، فإن الآلية الدقيقة لكيفية مساهمة IL-17 في تطور عدوى فيروس التهاب الكبد الوبائي لا تزال غير واضح.

هدف الدراسة: كان الهدف من هذه الدراسة هو فحص العلاقة بين الإصابة بفيروس التهاب الكبد الوبائي (HCV) و IL-17. تم ذلك عن طريق قياس مستويات الإنترلوكينات في الدم والحمل الفيروسي لفيروس التهاب الكبد الوبائي (HCV) لدى المرضى المصابين بعدوى فيروس التهاب الكبد الوبائي (HCV).

المنهجية: شملت الدراسة خمسين مريضا بفيروس التهاب الكبد الوبائي الذين كانوا يتلقون العلاج في مستشفى أمراض الجهاز الهضمي والكبد التعليمي / مجمع مدينة الطب في بغداد. أجريت الدراسة في الفترة ما بين نوفمبر 2022 وأبريل 2023، وتمت مطابقة مجموعات الدراسة مع 50 مجموعة ضابطة على أساس العمر والجنس. تم أخذ سبعة ملليلتر من عينات الدم الوريدي من جميع المشاركين لاختبار الأجسام المضادة لفيروس التهاب الكبد الوبائي باستخدام مجموعة ELISA، والقياس الكمي لـ HCV RNA بواسطة PCR، وتقدير مستويات المصل لـ IL-17.

النتائج: كان نطاق عيار الحمض النووي الريبي (RNA) الفيروسي لفيروس التهاب الكبد الوبائي (HCV) بين المرضى هو 165-55595000 نسخة/مل، وكانت جميعهم إيجابيين للأجسام المضادة لفيروس التهاب الكبد الوبائي (HCV). من ناحية أخرى، جاءت نتيجة اختبار المجموعة الضابطة سلبية للفيروس. كان متوسط تركيز IL-17 في مجموعة المرضى 1458.03 (±165.7) بيكوغرام/مل، بينما كان لدى المجموعة الضابطة متوسط 628.04 (±163.0) بيكوغرام/مل (P <0.0001). وكانت قيمة R للحمل الفيروسي HCV وIL-17 هي 0.1388. تجدر الإشارة إلى أن متوسط تركيز IL-17 في المصل كان الأعلى بين المرضى الذين تتراوح أعمارهم بين ≤ 50 عاما. أما بالنسبة لمرضى فيروس التهاب الكبد C الذين يتلقون العلاج، فقد بلغ متوسط الحمل الفيروسي لديهم 406,3051.90 نسخة/مل، وكان متوسط تركيز IL-17 لديهم 1444.56 بيكوغرام/مل. كان لدى المرضى الذين لم يتلقوا العلاج متوسط حمل فيروسي أعلى قدره 765,525.94 نسخة/مل، وكان متوسط تركيز IL-17 لديهم 1484.19 بيكوغرام/مل.

الخلاصة: تم العثور على تركيز IL-17 أعلى بكثير في المرضى الذين يعانون من فيروس التهاب الكبد الوبائي (HCV)، وأظهر ارتباطا إيجابيا ضعيفا مع الحمل الفيروسي لفيروس التهاب الكبد الوبائي. علاوة على ذلك، كان لدى المرضى الأكبر سنا المصابين بفيروس التهاب الكبد الوبائي مستويات أعلى من IL-17 في دمائهم. ومع ذلك، فإن قياس الحمل الفيروسي لفيروس التهاب الكبد الوبائي (HCV) ومستوى IL-17 لم يظهر أي اختلافات كبيرة في قراءاتهما.

الكلمات المفتاحية: IL-17، فيروس التهاب الكبد C، الانترلوكينات المرتبطة ب HCV، التهاب الكبد المزمن.