



1A and 4A Genotypes of Hepatitis C Virus and Their Influence on Some Biochemical and Hematological Changes in Infected Iraqi Patient

Dhuha Sabah Hasan^{1*}, Alea Farhan Salman¹, Muhana Oweed Hussein², Aseel Majeed Hameed¹, Fatma Abd Alhamza Obed¹, Sara thamer abed³, Yousef Rhamadhan Salman¹ and Adian hussien ali¹.

¹National Center of Hematology, Mustansiriyah University, Baghdad, Iraq.

²Middle Technical University, Al-Anbar Technical Institute, Al-Anbar, Iraq.

³Department of chemistry, College of Science, University of Diyala, Diyala, Iraq.

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ABSTRACT

Hepatitis C Virus (HCV) causes about 70 million chronic infections, posing a serious threat to global health. This study aimed to determine the most common HCV genotypes and their effects on liver enzyme and hematological parameter concentrations in Iraqi patients. This study examined 100 untreated HCV cases (infected Iraqi patients; age mean±SD: 41.99±16.78 years) and 100 uninfected HCV cases (control group; age mean±SD: 39.64±13.55 years). The study was conducted from January 2024 to October 2024 at Al-Yarmouk Teaching Hospital in Baghdad, Iraq. Viral genotypes were determined by real-time PCR. Statistical analysis showed a significant increase in hematological parameters, with hemoglobin and PLT levels being significantly higher in infected patients compared to the control group ($p < 0.05$). Similarly, liver enzymes were also increased in Iraqi patients infected with genotypes 1A and 4A of hepatitis C virus (HCV). Patients with the 4A genotype were older on average and showed a greater influence on laboratory parameters, possibly due to a weakened immune response associated with advanced age. In addition, Iraqi patients infected with HCV showed a higher prevalence of the 1A genotype (57%) compared to the 4A genotype (43%). This paper contributes to understanding the relationship between genotypes of HCV and changes in biochemical and hematological parameters by providing accurate information that helps in making evidence-based medical decisions that meet the patients' needs more accurately and effectively.

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Corresponding Author:

Dhuha Sabah Hasan

National Center of Hematology,
Mustansiriyah University,
Baghdad, Iraq

Email dhuha.sabah@uomustansiriyah.edu.iq



1. INTRODUCTION

Hepatitis C Virus (HCV) is a blood-borne infection that can cause mild to severe disease, including liver cirrhosis and hepatocellular carcinoma (HCC). It can lead to both acute and chronic hepatitis, and it's a global health issue, causing significant liver damage to around 70 million people worldwide [1]. Indicators of acute viral hepatitis include jaundice, elevated serum alanine aminotransferase (ALT, AST) activity, serum antibodies to HCV or RNA of HCV, and the presence of hepatitis C virus antigen (HCV antigen) within the first six months following HCV infection [2]. The high rate of spontaneous mutation in RNA-enveloped viruses results in a varied collection of viruses with varying nucleotide sequences. Numerous subtypes, including genotypes 1, 2, 3, 4, 5 and 6, are among the six main genotypes. Small letters are used to indicate subtypes, such as genotypes 1a and 1b [3]. Genotypes of HCV and subtypes vary in their geographic distributions and correlations with demographic risk categories [4].

Subtype 1b is also widespread in most European nations, whereas subtype 1a is more common in HIV-positive individuals. Genotype 1a is the most common type worldwide. The most prevalent subtype, however, is 1a throughout North America, portions of South America, the UK, Scandinavia, and Australia [5]. Hepatitis C virus genotype 4 (HCV-4) is considered to be the most virulent of the various genotypes. More than 80% of hepatitis C infections are caused by this kind of virus, which is most common in Africa and the Middle East. Hepatocellular carcinoma and cirrhosis, which can result in liver failure, are mostly caused by this infection [6, 7]. The type and duration of treatment for HCV are strongly influenced by genotype [8]. The most common ways of blood-borne HCV transmission are: Reusing or not properly sterilizing medical equipment, particularly syringes and needles, in healthcare institutions, giving blood and blood products without screening and using injection equipment to provide drugs [9, 10]. Sharing food or beverages with an infected person does not spread the virus from one person to another [11, 12]. At 15%, Egyptians have the highest global prevalence of HCV, with 90% of infections caused by 4A genotype of HCV. 62% of infections in Saudi Arabia are caused by 4A genotype of HCV, and the prevalence is 1-3 percent [13, 14]. Higher prevalence rates of 10–24% have been recorded for 4A genotype of HCV in several Mediterranean Sea-front nations in southern Europe, including France, Greece, Spain, and Italy [15]. By estimating the incidence of hepatitis C virus (HCV) genotypes 1a and 4a among other hepatitis C genes in Iraqi patients and determining the most common genotypes of HCV and their effect on some liver enzymes and hematological parameters. This study aims to provide insight into the common genotypes of HCV in the Iraqi community, allowing to better understand the relationship between genotypes of HCV and biochemical and hematological changes by providing information that helps in making evidence-based medical decisions because these genotypes have a significant effect on the type and duration of treatment used to treat HCV infection. The present study will also compare genotypes of HCV based on the age of the patient.

2. Ethical approval

The study received approval from all patients and healthy individuals at The National Center of Hematology, Mustansiriyah University, Baghdad, Iraq, and was also approved by the local ethics committee.

3. METHOD

The current study included 100 Iraqi patients with chronic hepatitis C virus (HCV) infection and 100 healthy controls. The sample was divided into two groups: the patient group, which included 100 patients with chronic HCV infection (48 males and 52 females), and the control group, which included 100 healthy individuals (50 males and 50 females). The two groups were matched in terms of weight, sex, and age, ranging in age from 18 to 66 years, and lived in the same region and environment. Patient and control samples were collected from patients attending the National Center for Hematology/Al-Mustansiriya University and Al-Yarmouk Teaching Hospital in Baghdad, Iraq, between January 2024 and October 2024. All patients and controls gave written informed consent to participate in this study, and prior approval was obtained from the Ethics Committee of the National Center for Hematology Research and Treatment. Patients were diagnosed with chronic liver disease (CHCV) for more than eight months, based on confirmatory nucleic acid testing (NAT) and hepatitis C virus (HCV) antibody testing. All control subjects underwent serological testing before enrollment in the trial, including HCV antibody testing, and the results were negative. Abdominal ultrasound was used to evaluate liver echocardiography, symptoms of portal hypertension, or radiographic signs of cirrhosis to rule out liver cancer. Serum AST and ALT were measured using a cobas (c111) analyzer (ROCHE, Germany). And CBC was measured using a NORMA Icon 3 blood analyzer. Additionally, a comprehensive clinical evaluation was performed. HCV antibodies was detected using an enzyme-linked immunosorbent assay (ELISA) using a commercial diagnostic kit (CTKBiotech, USA) designed for the detection of antigen in serum. HCV genotypes were determined using the polymerase chain reaction (PCR) technique using the primer-specific extension analysis (PSEA-HCV). The reaction program was carried out on a thermal cycler under the following conditions: initial denaturation at 94°C for 20 seconds, followed by 30 cycles of amplification, which included heating at 94°C for 1 minute, coupling at 54°C for 45 seconds, and extension at 72°C for 2 minutes. Adult patients of both sexes who were serologically positive for hepatitis C virus (HCV) antibodies met the inclusion and exclusion criteria. Inclusion criteria included: 1- Age \geq 18 years, 2- Confirmed diagnosis of chronic HCV infection, 3- Presence of disease activity (elevated ALT/AST), 4- Patient consent to participate. Exclusion criteria were: 1- Age < 18 years, 2- Advanced liver failure (Child-Pugh C) or serious complications, 3- Active or metastatic liver cancer, 4- Uncontrolled alcohol or drug use, 5- Pregnancy or breastfeeding.

Patients taking antiviral drugs, those with bacterial and autoimmune diseases, or with alcoholism and fatty liver, those or toxic hepatitis from specific medications and those with other forms of hepatitis were excluded. SPSS version 10.0 for Windows (SPSS, Chicago, Illinois, USA) was used for data collection and statistical analysis (Mann-Whitney U tests). In this regard, a P-value of less than 0.05 was deemed statistically significant.

4. RESULTS

Table 1: Baseline Demographic and Clinical Characteristics of HCV Patients and Control Groups.

Variable	Control group	Patient group	P-value
Number	100	100	
Age (year)	39.64±13.55	41.99±16.78	0.276
BMI (Kg/m ²)	25.52±3.47	25.56±5.06	0.909
Normal	39(39%)	27(27%)	
Overweight	45(45%)	35(35%)	
Obese	15 (15%)	25(25%)	
Gender M/F	50/50	52/48	0.777
Genotype strain of HCV	Ratio		
1A	57%		
4A	43%		

BMI: Body Mass Index, M: Male, F: Female, HCV: Hepatitis C Viruses.

Table2: Comparison between patients with HCV (1A genotype) and control group

Variable	Patient Mean± SD	Control Mean± SD	P-value
Age (year)	38.40±17.51	43.66±10.76	0.06
BMI (Kg/m ²)	25.50±5.19	25.72±2.91	0.91
WBCs (10 ⁹ /L)	6.96±1.88	7.21±0.85	0.28
PLT (10 ⁹ /L)	207.35±71.14	310.04±61.78	0.0001
Hb (g/dl)	9.81±1.86	13.55±0.94	0.0001
AST (U/L)	34.78±13.31	27.02±4.43	0.01
ALT (U/L)	36.95±17.36	24.01±5.82	0.001

Significant P < 0.05; WBCs: White Blood Cells, Hb: Hemoglobin, PLT: Platelet, AST: Aspartate transaminase, ALT: Alanine aminotransferase

When comparing patients with genotype 1A hepatitis B virus (HCV) with the control group (Table 2), a statistically significant difference was found in the levels of the liver enzymes AST and ALT, with p-values of 0.01 and 0.001, respectively. The results also showed that HCV patients were more likely to develop anemia than the control group, which did not exhibit this condition, with a high statistical significance (p = 0.0001). No statistically significant difference was found between patients with HCV and the control group (P-value = 0.28) in the mean value of WBCs, which were 6.96±1.88 and 7.2±0.85, respectively. The PLT mean in patients with HCV was 207.35±71.14 10⁹/L, while the PLT mean of the control group was 310.04±61.78 10⁹/L, indicating a statistically significant difference between them (P-value = 0.0001). There were no statistically significant differences in age and BMI.

Table 3: Comparison between patients with HCV (4A genotype) and control group

Variable	Patient Mean± SD	Control Mean± SD	P-value
Age (year)	46.74± 14.62	44.6±10.71	0.81
BMI (Kg/m ²)	26.0±5.11	25.0± 2.90	0.464
WBCs (10 ⁹ /L)	6.8±2.32	7.2±0.85	0.28
PLT (10 ⁹ /L)	185.04±83.51	319.04±61.78	0.0001
Hb (g/dl)	10.5±1.95	13.55±0.94	0.0001
AST (U/L)	41.33±14.69	27.0±4.43	0.0001
ALT (U/L)	49.80±13.66	24.0±5.82	0.0001

Significant P < 0.05; WBCs: White Blood Cells, Hb: Hemoglobin, PLT: Platelet, AST: Aspartate transaminase, ALT: Alanine aminotransferase.

Table 3 showed a statistically significant increase in AST and ALT levels in patients with the 4A genotype of HCV compared to the control group (P-value = 0.0001 and 0.0001), respectively. Patients with the 4A genotype of HCV were found to be anemic compared to the control group who were found to be free of anemia with Hb levels of 10.5 ± 1.95 g/dL and 13.55 ± 0.94 g/dL, respectively. Therefore, there was statistical significance between the two groups of patients and control (P-value = 0.0001). There was no statistically significant difference between both groups in WBCs mean (P-value = 0.28). The PLT mean in patients with HCV was 185.04 ± 83.51 109/L, while the PLT mean of the control group was 319.04 ± 61.78 109/L, indicating the presence of statistical significance between them (P-value = 0.0001). There were no statistically significant differences in age and BMI.

Table 4: Comparison between 1A and 4A genotypes of HCV according to their age

Genotype	mean \pm SD	P-value
1A	38.40 \pm 17.51	0.013
4A	46.74 \pm 14.62	

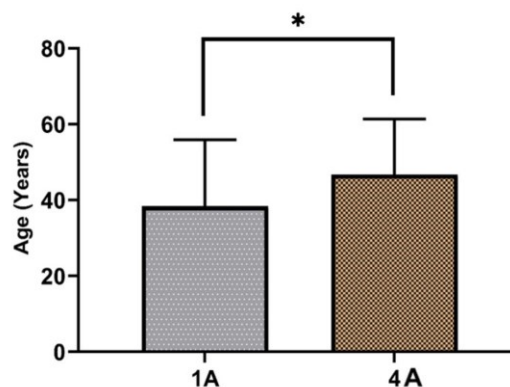


Figure 1. Comparison of 1A and 4A genotypes of HCV and age.

The present study shows statistically significant differences in genotypes of HCV according to their age between patients infected with 1A genotype of HCV (mean \pm SD = 38.40 \pm 17.51) and patients infected with 4A genotype of HCV (mean \pm SD = 46.74 \pm 14.62) with a statistical value (P-value = 0.013), as shown in Figure 1 and Table 4.

5. DISCUSSION

Determining the origin of an epidemic and infection requires an understanding of viral genotypes. HCV can spread vertically (from mother to child) or horizontally (from person to person), as well as through needle injections or intercourse. Several studies have also demonstrated the unique pathogenicity of HCV subtypes. Given the usual mechanisms of HCV transmission, one genotype may be more prevalent than the others that have been documented [16]. This may be the first study to focus on the clinical and virological features of HCV infection in Baghdad, Iraq, as well as the genetic diversity of HCV. 57% of HCV infections were of 1A genotype, while 43% of participants in this study were 4A genotype. The present results are comparable to other studies conducted in an Iraqi sample from Baghdad and reported by author Sahib A. Hussein. The study observed 1A and 4A genotypes of HCV, with the latter being the most prevalent. Globally, 1A genotype of HCV is the most common at 46% and is also found in Europe [17-18]. The Middle East and Africa regions account for the majority of 4A genotype of HCV infection [19]. In addition to being crucial to the development of a successful vaccine, determining the genotype of HCV within a certain population is a major problem in investigating the evolution of HCV infection in different geographical locations [20-21].

According to the results shown in Tables 2 and 3, it can be observed that the hematological parameters (such as Hb and PLT) were significantly affected by 1A and 4A genotypes of HCV. Meanwhile, the WBCs showed a slight decrease, which was not statistically significant. The reduction of hematological parameters, especially Hb and PLT, indicates the presence of chronic HCV, which may be due to the direct viral effect or immune disorders. Chronic HCV can also affect the activity of the bone marrow, leading to a decrease in the production of blood cells. Although hematological parameters may indicate the progression of HCV disease, they are not conclusive evidence alone, as other factors, such as elevated liver enzymes, must be considered.

The liver enzymes in patients infected with the 1A genotype of HCV showed significant differences between the two groups of patients and the control (see Table 2). On the other hand, patients infected with the 4A genotype of HCV had higher levels of liver enzymes (AST and ALT) than patients infected with the 1A genotype of HCV, as well as compared to the control group. This increase is likely due to several factors, including the interaction of multiple pathological mechanisms, including the severity of inflammation, the rate of fibrosis, viral activity, the intensity of the immune response, and concomitant epidemiological factors. These differences highlight the importance of genotype in determining the severity of infection and disease progression, which must be taken into account during diagnosis and therapeutic evaluation. This is consistent with prior research investigating the relationship between HCV subtypes and liver disorders. Patients with the 4A genotype of HCV were more susceptible to develop cirrhosis (45.2%) than those with the non-4A genotype of HCV (13.3%) [22]. According to earlier research conducted in central Iraq, 50% of recruited samples had the 1A genotype and 35% of samples had the 4A genotype [23]. In this study, 2 and 3 genotypes were not revealed, while in another study, 53% of samples were categorized as HCV genotype 4, with 23% being genotype 1, 20% being genotype 3 and 3% being genotype 2. According to a previous study conducted in Iran, the majority of Iranian patients with chronic HCV infection were infected with genotype 1 (47%) and genotype 3 (36%) [14]. In other studies, genotype 4 was the most common HCV genotype in Saudi Arabia, Kuwait [25] and Yemen [26]. This variance is likely to be explained by variations in race, due to the geographical connectivity with the region (especially Egypt and the Middle East), unsafe historical and medical transmission routes, in addition to the ability of these two types to persist and adapt [26]. The distribution of HCV infections may be influenced by population age. For instance, subtype 3b was the most prevalent subtype in younger people (10–20 years old), whereas subtypes 1a and 1b were more prevalent in older patients (51–60 years old) [27]. Several studies have demonstrated a correlation between HCV genotypes and age distribution. The results of the present study showed that the prevalence of HCV infection varied by age and was higher in patients aged 46 years and older, as shown in Figure 1 and Tables 2, 3 and 4. This is consistent with other research that found that the prevalence of HCV infection was higher in older adults (over 45), and it was twice as high as that reported for the Iraqi population as a whole [28]. The age distribution can be attributed to genotypes of HCV as we performed HCV genotyping. Patients who acquire the virus in their later years have a faster and more frequent progression to cirrhosis [29–30].

6. CONCLUSION

The present study concluded that the prevalence of 1A genotype of HCV in Iraqi patients was higher than the 4A genotype of HCV, with a prevalence rate of about 57%. Hematological parameters, which were decreased in patients compared to the control group, especially hemoglobin and PLT, were affected by HCV genotypes 1A and 4A due to direct viral action or immune disorders that limit blood cell production. Patients infected with 4A genotype of HCV had elevated concentrations of liver enzymes (AST and ALT) compared to 1A genotype of HCV. Because patients infected with 4A genotype of HCV are older, making them more susceptible to complications such as cirrhosis due to their weak immune response. Moreover, 4A genotype of HCV may lead to more severe reactions compared to 1A genotype of HCV.

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REFERENCES

- [1] R. TH. Françoise, Epidemiology of hepatitis C virus infection, Clinics and Research in Hepatology and Gastroenterology, Volume 45, Issue 3, 2021, 101596, ISSN 2210-7401, <https://doi.org/10.1016/j.clinre.2020.101596>.
- [2] C. Gan, Y. Yuan, H. Shen, et al. Liver diseases: epidemiology, causes, trends and predictions. Sig Transduct Target Ther 10, 33 (2025). <https://doi.org/10.1038/s41392-024-02072-z>.
- [3] T.G.Villa, A.G. Abril, S. Sánchez, et al. Animal and human RNA viruses: genetic variability and ability to overcome vaccines. Arch Microbiol 203, 443–464 (2021). <https://doi.org/10.1007/s00203-020-02040-5>.
- [4] L. Lin, G. Wang, L. Hao, T. Yan, Genotype Distribution and Migration Patterns of Hepatitis C Virus in Shandong Province, China: Molecular Epidemiology and Phylogenetic Study JMIR Med Inform 2025. <https://doi.org/10.2196/60207>.
- [5] M. A. Daw, A. El-Bouzedi, & A. A. Dau, Geographic distribution of HCV genotypes in Libya and analysis of risk factors involved in their transmission. BMC Res Notes 8, 367 (2015). <https://doi.org/10.1186/s13104-015-1310-x>.
- [6] R. Shah, L. Ahoegbe, M. Niebel, J. Shepherd, EC. Thomson. Non-epidemic HCV genotypes in low- and middle-income countries and the risk of resistance to current direct-acting antiviral regimens. J Hepatol. 2021 Aug;75(2):462-473.. Epub 2021 May 8. <https://doi.org/10.1016/j.jhep.2021.04.045>.
- [7] E. Vo-Quang, J'Unusual . Pawlotsky, HCV genotype subtypes: origin, distribution, sensitivity to direct-acting antiviral drugs and behaviour on antiviral treatment and retreatment Gut 2024. PMID: 38782565; PMCID: PMC11347264. <http://doi:10.1136/gutjnl-2024-332177>.
- [8] T. Stroffolini, G. Stroffolini. Prevalence and Modes of Transmission of Hepatitis C Virus Infection: A Historical Worldwide Review. Viruses. 16(7):1115.. PMID: 39066277; PMCID: PMC11281430. 2024 Jul 11. <http://doi:10.3390/v16071115>.
- [9] D. Toma, L. Anghel, D. Patraş, & A. Ciubară, Hepatitis C Virus: Epidemiological Challenges and Global Strategies for Elimination. Viruses, 17(8), 1069. (2025). <https://doi.org/10.3390/v17081069>.
- [10] S.K. Stoszek, M. Abdel-Hamid, S. Narooz, M. El Daly, D. Saleh, N. Mikhail, et al. Prevalence of and risk factors for hepatitis C in rural pregnant Egyptian women. Trans R Soc Trop. Med. Hyg. 100:102–107.Epub 2005 Nov 9. PMID: 16289168. 2006. <http://doi:10.1016/j.trstmh.2005.05.021>.

- [11] A. Gomaa, M. Gomaa, N. Allam, I. Waked, Hepatitis C Elimination in Egypt: Story of Success. *Pathogens* 2024, 13, 681. <https://doi.org/10.3390/pathogens13080681>.
- [12] H.P. Kattel, S. Sharma, K. Alfsnes, J.H.-O. Pettersson, R. Pathak, S.B. Engebretsen, K.R. Rijal, P. Ghimire, A.K. Andreassen, M.R. Banjara, The Genotypes/Subtypes and Antiviral Drug Resistance of the Hepatitis C Virus from Patients in a Tertiary Care Hospital in Nepal. *Viruses* 2025, 17, 377. <https://doi.org/10.3390/v17030377>
- [13] A. Elkady, Y. Tanaka, F. Kurbanov, F. Suguchi, M. Sugiyama, A. Khan, Genetic variability of hepatitis C virus in South Egypt and its possible clinical implication. *J. Med. Virol.* 81 (6): 1015–1023. PMID: 19382263. 2018. <http://doi:10.1002/jmv.21492>.
- [14] I. Nazir, and E. Mohammed, Changing pattern of hepatitis viral infection in Saudi Arabia in the last two decades. *Journal of Hepatology*; 54: 1250–1262. PMID: 16868370. 2018. <http://doi:10.5144/0256-4947.2003.367>.
- [15] J. Esteban, S. Saulede, and J. Quer, The changing epidemiology of hepatitis C virus infection in Europe. *J. Hepatol.* 48:148–162. Epub 2007 Nov 5. PMID: 18022726. 2008. <http://doi:10.1016/j.jhep.2007.07.033>.
- [16] J.F. Quarleri, M.V. Bussy, V. L Mathet, V. Ruiz, R. Iacono, L. Lu, vitro detection of dissimilar amounts of hepatitis C virus (HCV) subtype-specific RNA genomes in mixes prepared from sera of persons infected with a single HCV genotype. *J. Clin. Microbiol.* 41:27272733. PMID: 12791916; PMCID: PMC156549. 2018. <http://doi:10.1128/JCM.41.6.2727-2733.2003>.
- [17] A. Sahib Hussein, H. Raghad Al-azzawi, Duraid Qassim Alshareef. Assessment of hepatitis C viral load and genotypes by molecular determination in Iraqi patients treated with Ledipasvir/sofosbuvir (Harvoni) drug. *Iraqi Journal of Science*, 2021, Vol. 62, No. 5, pp: 1431-1437. <https://doi.org/10.24996/ij.s.2021.62.5.5>
- [18] M. B. Sergio, H. Charlotte, P. Bandita, H. Robert, M. S. Luisa, M. B. Diana, G. S. Mani, G. M. John, M. E. S. Hongmei, and D. S. Stephen, Identification of a Novel Hepatitis C Virus Genotype from Punjab, India - Expanding Classification of Hepatitis C Virus into 8 Genotypes 2018. PMID: 29982508. <http://doi:10.1093/infdis/jiy401>.
- [19] H. Charlotte, P. Bandita, C. Silvia, Z. Stefan, M. Christophe, D. S. Stephen, M. B. Sergio, A. Tarik, A. Laurent, A. Armand, C. Jyh-Jou, C. Jane, K. Dharmesh, H. Robert, S. Peter, M. Hongmei, and S. S. Evguenia, Identification of 19 Novel Hepatitis C Virus Subtypes Further Expanding HCV Classification 2019. <https://doi:10.1093/ofid/ofz076>.
- [20] OG. Pybus, MA. Charleston, S. Gupta, A. Rambaut, EC. Holmes, PH. Harvey, The epidemic behavior of the hepatitis C virus. *Science*. 2001;292:2323–2325. PMID: 11423661. <http://doi:10.1126/science.1058321>.
- [21] JF. Cantaloube, P. Gallian, H. Attoui, P. De Biagini, P de Micco, X. Lamballerie, Genotype distribution and molecular epidemiology of hepatitis C virus in blood donors from southeast France. *J Clin Microbiol.* 43:3624–3629. PMID: 16081888; PMCID: PMC1233931. 2005. <https://doi.org/10.1128/jcm.43.8.3624-3629.2005>.
- [22] SC. Ray, RR. Arthur, A. Carella, J. Bukh, DL. Thomas, Genetic epidemiology of hepatitis C virus throughout Egypt. *J Infect Dis.* 182(3):698–707. Epub 2000 Aug 17. PMID: 10950762. 2000. <https://doi.org/10.1086/315786>.
- [23] W.A. Al Kubaisy, K.T. Al Naib, & M. Habib, (2006). Seroprevalence of hepatitis C virus specific antibodies among Iraqi children with thalassaemia. *EMHJ - Eastern Mediterranean Health Journal*, 12 (1-2), 204-210, 2006. <https://iris.who.int/handle/10665/117072>
- [24] K. Samimi-Rad. Molecular epidemiology of hepatitis C virus in Iran as reflected by phylogenetic analysis of the NS5B region. *Journal of Medical Virology*. 74: 246–252. PMID: 15332273. 2004. <https://doi:10.1002/jmv.20170>.
- [25] A. Koshy. Treatment of hepatitis C virus genotype 4-related cirrhosis: ribavirin and interferon combination compared with interferon alone. *Journal of Clinical Gastroenterology* 35:82–85. PMID: 12080232. 2002. <http://doi:10.1097/00004836-200207000-00017>.
- [26] N. R. Hussein, I. Tunjel, Z. basharat, A. taha and W. Irving. The treatment of HCV in patients with haemoglobinopathy in Kurdistan Region, Iraq: a single centre experience. *Epidemiol. Infect.* 144, 1634–1640. PMID: 27125573; PMCID: PMC9150703. (2016). <http://doi:10.1017/S0950268815003064>.
- [27] Z. Niu, P. Zhang, Y. Tong. Age and gender distribution of hepatitis C virus prevalence and genotypes of individuals of physical examination in Wuhan, Central China. *Springerplus*. 2016. <https://doi.org/10.1186/s40064-016-3224-z>.
- [28] M. AL. Abdulghani, AB. Gailan, and H. AL. Amina, Seroprevalence of Hepatitis C Virus in Iraqi Population. *JOJ Immuno Virology*; 2016;1(3) 1557. <http://doi:10.1186/s40064-016-3224-z>.
- [29] F. Cainelli, Hepatitis C virus infection in the elderly: epidemiology, natural history and management. *Drugs Aging*. 2008;25(1):9-18. <http://doi:10.2165/00002512-200825010-00002>.
- [30] A.A.Eham, AB. H. Ali, F. S. Alea. Serum Elabela Level as a Reliable Biomarker for Predicting of Liver Fibrosis in Iraqi Patients with Chronic Hepatitis C. *Medical Journal of Babylon*. Vol 21; Issue 2;346-352. 2024. http://doi:10.4103/MJBL.MJBL_666_23.

BIOGRAPHIES OF AUTHORS

	<p>Asst. lect. Dhuha Sabah Hasan is Assistant lecturer at the National Center for Hematology, Al-Mustansiriya University, Baghdad, Iraq. She holds a Bachelor of Science degree in Chemistry from the University of Diyala/College of Science, and a Master of Science degree in Chemistry/Materials Chemistry from the Polytechnic University of Romania. Her research areas include biomaterials, biochemistry, and materials science. She has published numerous scientific papers in local and international conferences and journals. She can be contacted via email at: dhuha.sabah@uomustansiriyah.edu.iq</p>
	<p>Scopus®  </p>
	<p>Dr. Aliyah Farhan Salman is an Assistant Professor at the National Center for Research and Treatment of Blood Diseases at Al-Mustansiriya University, Iraq. She holds a Bachelor's degree in Chemistry and a Master's degree from the University of Baghdad, Iraq. She also holds a PhD in Clinical Chemistry from the College of Medicine at Al-Mustansiriya University. She has published numerous scientific papers in local and international conferences and journals. She can be contacted via email at aliyah_farhan@uomustansiriyah.edu.iq.</p>
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