Comparison of Three Different Treatment Regimens of HCV Infection in 295 Iraqi Patients

DOIs: https://doi.org/10.32007/66011-17

Wasan K. Jasim AL-Saedi* FIBMS
Nawal M. Firhan AL-Khalidi* CABM, FICMIS
Ahmed A. Hussein ** PhD

Abstract:

**Background:** Viral Hepatitis C infection is global public health problem throughout the world. Different treatment regimens are used which produce different rates of response affected by many factors.

**Objectives:** To assess the efficacy of three different treatment regimens in 295 Iraqi patients infected with chronic HCV.

**Patients and methods:** This is an observational cohort study; in which 295 (133 male and 162 female) patients with chronic HCV infection were enrolled during the period between August 2015 to January 2017 from Gastroenterology Clinic of Baghdad Teaching Hospital and Gastroenterology and Hepatology Teaching Hospital. Baseline HCV viral load measurements and genotyping were done for each patient. Patients were followed up by viral load measurement at end of the treatment period and three months after the end of the treatment.

**Results:** The majority of patients infected with chronic HCV achieved sustained virological response (SVR) (defined as undetectable HCV RNA 12 to 24 weeks after the end of the treatment); were of the generic sofosbuvir/ledipasvir treatment group (51 out of 72 (70.8%) followed by generic Sofosbuvir with (peg interferon/ribavirin) treatment group (68 out of 111 (61.3%) followed by (peg interferon/ribavirin) treatment group (42 out of 86 (48.8%).

**Conclusions:** The best treatment efficacy was obtained with generic sofosbuvir/ledipasvir followed by sofosbuvir with peg interferon and ribavirin then peg interferon and ribavirin. The most responder genotype in Iraqi patients was genotype 4 and the least responder genotype was genotype 1b.

**Key words:** HCV, sofosbuvir, ribavirin, INF

Introduction:

Hepatitis C is an infectious disease caused by hepatitis C virus which is a single strand ribonucleic acid (RNA) virus affecting initially the liver, and may lead to hepatic and extra hepatic complications. The virus can cause both acute and chronic hepatitis infection ranging in severity from a mild illness lasting a few weeks to a serious lifelong illness. (1) Hepatitis C virus includes six major genotypes which determine the type, duration of and response to treatment. Several drugs were developed to treat HCV infection beginning with the use of interferon, addition of ribavirin, using of pegylated forms of interferon and addition of direct antiviral drugs. Recently the HCV treatment guidelines directed towards the use of an interferon free regimens to avoid adverse effects associated with the interferon use. (2) The aim of treatment of HCV infection is to cure the infection, which is measured by sustained virological response (SVR) (defined as undetectable HCV RNA 12 weeks to 24 weeks after the end of treatment). Achieving SVR associated with the decrease of hepatic and extra hepatic complications, prevents the spread of the infection and improves the quality of life. (3) New drugs for HCV infection were used to treat Iraqi patients (including sofosbuvir and sofosbuvir/ledipasvir combination). These drugs are of generic origins. There is need to study the efficacy of these drugs in the treatment of HCV infected Iraqi patients.

Patients and Methods:

**Study design and settings:** This is a comparative study of three treatment regimens conducted at the Gastroenterology and Hepatology Teaching Hospital...
and Baghdad Teaching Hospital of the medical city – Baghdad - Iraq from August 2015 to January 2017. The study included 295 naïve adult patients (133 males and 162 females) with chronic HCV infection (over 18 years of age).

**Exclusion criteria were:**
Conditions related to the liver: acute infection; co-infection with other viruses (HBV or other liver infecting viruses); liver cirrhosis; or liver transplanted patients, immune system conditions; patients using immune modulator drugs like systemic steroid, interferon, interleukins or cytokines; patients with autoimmune diseases; or immunocompromised, patients with co-morbidity: Renal impairment; malignancy; or thalassaemia, conditions related to the treatment: patients who had no measurement of viral load before and at the end of treatment; patients who stopped treatment or were non-compliant because of side effects or due to any other cause; or patients who failed on previous treatment regimens and others: Pregnant women; or alcoholic patients.

**Patients:**
Two hundred ninety five patients with chronic HCV were enrolled in this study, and were randomly distributed by a specialist physician into three groups according to the treatment regimen they received, taking into consideration the cost and availability of the drugs. In August 2015, two regimens were used, which were:

1. Peginterferon with ribavirin group (PR): 92 HCV infected patients who underwent PR treatment for 48 week of peg-IFN - alfa-2a prefilled syringe subcutaneously in a dose of 180 mcg once a week or peg-IFN - alfa-2b prefilled syringe subcutaneously in a dose of 1.5mcg/kg/week with ribavirin 200 mg capsule in a dose calculated according to body weight: Those ≥ 75kg received 1200mg daily and those < 75 kg received 1000mg daily. Both peg interferon and ribavirin might be given in dose modification later on according to their adverse effects. The study began in 2015 and the guidelines at that time permit use of PR regimen.

2. Peginterferon with ribavirin and sofosbuvir group (SOF+PR): 125 HCV infected patients were given peginterferon with ribavirin in a dose similar to first group with addition of sofosbuvir 400mg tablet daily for 12 weeks period. Then in 2016 sofosbuvir with ledipasvir were prescribed to HCV infected patients who were included later in this study.

3. Sofosbuvir and ledipasvir group (SOF/LDV): 78 HCV infected patients were given sofosbuvir 400mg and ledipasvir 90 mg as a single tablet per day for 12 weeks.

Patients were followed up for 48 weeks for the first regimen and for 12 weeks for second and third regimens by the measurement of viral load before and at the end of the treatment (ETR), and 3 months after the end of treatment (SVR). Twenty six patients were lost to follow up by SVR, for different reasons (6 patients of PR group, 14 patients of SOF+PR group and 6 patients of SOF/LDV group).

**Ethical consideration:** Participants agreed to participate in this study after being prepared to understand the aims of the study. This study was submitted to Iraqi Board for Medical Specializations / Clinical Pharmacy Board.

**Sample collection and processing:** Five milliliters blood samples were collected from patients suspected of having HCV in sterile test tubes. The samples were processed and analyzed in the serology section of the G.I.T Center at the Medical City Teaching Hospital. The sera were separated and screened for HCV antibodies by using HCV ELISA test kit which utilizes antigens from the core, NS3, NS4, and NS5 regions of the virus. Antigens have been carefully developed and selected to provide a sensitive and specific diagnostic test. Positive serum was stored in (-20°C) unit test for viral load. HCV viral load measurement was done in the private Nursing Home Hospital and Gastroenterology and Hepatology Hospital in the Medical City and in Dubai private laboratory while genotyping was done in Dubai private laboratory only. HCV detection by polymerase chain reaction (PCR) is based on the amplification of specific sites of the pathogen genome. In real-time PCR the amplified product is detected by fluorescent dyes. These are usually linked to oligonucleotide probes that bind specifically to the amplified product. Monitoring of fluorescence intensities during the PCR run (i.e., in real time) permits the detection and quantitation of accumulating product without having to re-open the reaction tubes after the PCR run. HCV RNA viral load was determined following the manufacturers recommendations by a sensitive PCR based assay (COBAS amplicor; ROCHE diagnostic).Kit: COBAS®AmpliPrep/ COBAS TaqMan® HCV Quantitative test, v2.0) manufactured by Roche /Germany. HCV genotyping test was done based on reverse-hybridization standard; biotinylated amplicons, generated by RT-PCR of the 5,UTR and Core regions of HCV RNA, are hybridized to specific probes that are bound to nitrocellulose strip by a polymer-T tail; biotinylated hybrids are then detected using streptavidin bound to alkaline phosphatase; amplicons that are not complemented are washed out. Then the substrate reacts with the streptavidin-alkaline phosphatase complex forming purple precipitate and coloring banding pattern on the strip (the instrument used was Rotor-Gen Q manufactured by Qiagen Hiden-Germany and the kit used was (GEN-C 2.0 manufactured by nuclear laser medicine S.r.i (Italy)). Although measurement of viral load is important after one month of treatment, the study does not include this measurement in order to reduce the cost for the patients.
Medication manufacturing origin: PR treatment regimen were 100% (n=92) of American origin in this study while sofosbuvir in SOF/PR regimen 50.4% (n=63) were from Indian origin and 44.8% (n=56) were from Egyptian origin; SOF/LDV regimen 84.5% (n=65) were of Indian origin and 11.3% (n=9) were of Egyptian origin.

Statistical analysis: Each patient was assigned a serial identification number. The data were analyzed using Statistical Package for Social Sciences (SPSS) version 19. The continuous data were represented by median and inter quartile range. The categorical data presented as frequency and percentage tables; Binary logistic regression was used to assess the association. Continuous data were tested for normality by Kolmogorov – Smirnov test. Non-normally distributed data were analyzed using nonparametric tests (Mann-Whitney U test, Median test, Wilcoxon test).

Categorical data were analyzed using the Chi-square test; P – value less than 0.05 was used as the alpha level of significance.

End point: Compare sustained virological response of the three different regimens

Results:

Baseline characteristics of the study population:
Female represented 51.1% of the PR group, 51.2% of the SOF+PR group, while in SOF/LDV group they represented 65.4%, but this was statistically non-significant. The median age of patients in the three groups did not differ significantly which was: 36 years for PR group, 39 years for SOF+PR and 38 years for SOF/LDV. Moreover, viral load measurements were not statistically different at baseline. Laboratory test including (White Blood Cells, platelets, granulocytes, aspartate aminotransferase (AST), alanine aminotransferase (ALT), Alkaline phosphatase (ALK), Albumin, Total serum. bilirubin (TSB), international normalized ratio (INR), Random blood sugar (RBS), Blood urea, and Serum creatinine) were not statistically significantly different, except for the hemoglobin (p value 0.017) which was statistically significantly different (Table 1(a),(b)).

Table 1(a): Baseline characteristic of categorical variables of the study population

<table>
<thead>
<tr>
<th>Variable</th>
<th>PR</th>
<th>SOF+PR</th>
<th>SOF/LDV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Viral load &lt;800000 IU/mL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Viral load ≥800000 IU/mL</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Chi-square test (P-value significant at alpha<0.05)
PR: PEG-IFN-alfa-2a or alfa-2b + ribavirin,
SOF + PR: Sofosbuvir + peg-IFN-alfa-2a or alfa-2b + ribavirin,
SOF/LDV: Sofosbuvir + ledipasvir

Table 1(b): Baseline characteristic of continuous variable of the study population

<table>
<thead>
<tr>
<th>Variable</th>
<th>PR</th>
<th>SOF+PR</th>
<th>SOF/LDV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>Median (IQR)</td>
<td>Median (IQR)</td>
<td>Median (IQR)</td>
</tr>
<tr>
<td>WBC*10^3/µL</td>
<td>5.1 (4.3-6.9)</td>
<td>6.3 (4.8-7.5)</td>
<td>6 (4.1-8)</td>
</tr>
<tr>
<td>Hb (g/dl)</td>
<td>12.7 (11.5-14.5)</td>
<td>13.5 (12.1-15)</td>
<td>12.7 (11.7-14.5)</td>
</tr>
<tr>
<td>Platelet*10^3/µL</td>
<td>211 (166-268)</td>
<td>239 (179-305)</td>
<td>197 (150-252)</td>
</tr>
<tr>
<td>Granulocyte*10^3/µL</td>
<td>3 (1.95-3.95)</td>
<td>3.7 (2.6-4.6)</td>
<td>3.2 (2.2-4.7)</td>
</tr>
<tr>
<td>Albumin g/dl</td>
<td>4.1 (3.85-4.5)</td>
<td>4 (3.5-4)</td>
<td>3.9 (3.7-4.1)</td>
</tr>
<tr>
<td>AST (iu/l)</td>
<td>36 (26-56)</td>
<td>37 (27-67)</td>
<td>41 (24-63)</td>
</tr>
<tr>
<td>ALT (iu/l)</td>
<td>40.5 (25-74)</td>
<td>47 (33-69)</td>
<td>46.7 (24-83)</td>
</tr>
<tr>
<td>ALK (iu/l)</td>
<td>82 (62-105)</td>
<td>87 (67-104)</td>
<td>87 (67-108)</td>
</tr>
<tr>
<td>TSB (mg/dl)</td>
<td>0.7 (0.5-0.97)</td>
<td>0.6 (0.5-0.9)</td>
<td>0.71 (0.59-1.17)</td>
</tr>
<tr>
<td>INR</td>
<td>1 (1-1.1)</td>
<td>1 (0.9-1.03)</td>
<td>1 (1-1.2)</td>
</tr>
<tr>
<td>Urea (mg/dl)</td>
<td>27 (22-33)</td>
<td>23.5 (20-30)</td>
<td>27.7 (22-34)</td>
</tr>
<tr>
<td>S.cr (mg/dl)</td>
<td>0.7 (0.69-0.81)</td>
<td>0.7 (0.6-0.8)</td>
<td>0.8 (0.7-0.9)</td>
</tr>
</tbody>
</table>

*Median test
IQR interquartile range (P-value significant at alpha <0.05)
PR: PEG-IFN-alfa-2a or alfa-2b + ribavirin
SOF+PR: Sofosbuvir + peg-IFN-alfa-2a or alfa-2b + ribavirin
SOF/LDV: Sofosbuvir + ledipasvir

HCV genotype 4 was present in 48.5% (n=133) of cases followed by genotype 1a in 22.3% (n=61), genotype 1b in 21.5% (n=59), non-specified genotype 1 subtype in 6.2% (n=17) and genotype 1a/b in 0.4% (n=1), so total subtypes of genotype 1 represented 50.4% (n=138) of
the cases. Genotypes 3 was present in 0.4% (n=1) and genotype 6 was present in 0.7% (n=2).

At the end of treatment, 80% (n=100) of patients responded to SOF/PR regimen; 75.6% (n=59) of patients respond to SOF/LDV; 63% (n=58) of patients respond to PR, which was statically significance (p-value=0.001). The results show that 70.8% (n=51) of patient on SOF/LDV regimen; 61.3% (n=68) of patients on SOF/PR regimen and 48.8% (n=42) of patients on PR achieved SVR, which was statically significance (p-value=0.018) figure (1).

By comparing the responders who achieved SVR to non-responder of all types of treatment, we found that the use of SOF/LDV is associated with a significant response to treatment compared to the other treatment regimens, with more than a three-fold increase in response to treatment (odds ratio = 3.143; 95% CI = (1.591 - 6.208); p=0.001). Genotype 4 is associated with a better response to treatment, with nearly a two-fold increase in response to treatment compared to other genotypes and it was statistically significant (odds ratio = 1.959; 95% CI = (1.079 - 3.558); p=0.001). The most responder genotype to PR was genotype 4 in 53.2% (n=25) of patients affected by G 4 followed by genotype 1a in 46.7% (n=7) of patients affected by genotype 1a, then genotype 1b in 40% (n=6) PR of patients affected by genotype 1b; but these differences were statistically non significance (p-value=0.300). The most responder genotype to SOF/PR was genotype 4 in 68.2% (n=30) of patients affected by genotype 4 followed by genotype 1a in 63.6% (n=14) of patients affected by genotype 1a, then genotype 1b in 50% (n=11) of patients affected by genotype 1b, but these differences were statistically not significance (p-value=0.248). The most responder genotype to SOF/LDV was genotype 4 in 74.2% (n=23) of patients affected by G 4 followed by G 1a in 68.4% (n=13) of patients affected by G 1a, then G 1b at 58.8% (n=10) of patients affected by G 1b, but these differences were statistically not significance (p-value=0.249) figure (2).

By comparing the responders who achieved SVR to non-responder of all types of treatment, we found that the use of SOF/LDV is associated with a significant response to treatment compared to the other treatment regimens, with more than a three-fold increase in response to treatment (odds ratio = 3.143; 95% CI = (1.591 - 6.208); p=0.001). Genotype 4 is associated with a better response to treatment, with nearly a two-fold increase in response to treatment compared to other genotypes and it was statistically significant (odds ratio = 1.959; 95% CI = (1.079 - 3.558); p=0.001). The most responder genotype to PR was genotype 4 in 53.2% (n=25) of patients affected by G 4 followed by genotype 1a in 46.7% (n=7) of patients affected by genotype 1a, then genotype 1b in 40% (n=6) PR of patients affected by genotype 1b; but these differences were statistically non significance (p-value=0.300). The most responder genotype to SOF/PR was genotype 4 in 68.2% (n=30) of patients affected by genotype 4 followed by genotype 1a in 63.6% (n=14) of patients affected by genotype 1a, then genotype 1b in 50% (n=11) of patients affected by genotype 1b, but these differences were statistically not significance (p-value=0.248). The most responder genotype to SOF/LDV was genotype 4 in 74.2% (n=23) of patients affected by G 4 followed by G 1a in 68.4% (n=13) of patients affected by G 1a, then G 1b at 58.8% (n=10) of patients affected by G 1b, but these differences were statistically not significance (p-value=0.249) figure (2).

Discussion:
Treatment of HCV infection and achieving SVR at which the patient is considered to be cured is a very important issue as it reduces the spread of infection, improves the quality of life and decreases the progression of disease.(4,5) The study aimed to evaluate the efficacy of the three types of treatment of hepatitis C infection in a group of Iraqi patients. The predominant genotype was genotype 4 (48.5% (n=133) followed by genotype 1a (22.3% (n=61); genotype 1b (21.5% (n=59)); and non-specified genotype 1 (6.2% (n=17). This result was similar to the results of Al-Kubaisy et al., (2015) in Iraq, who they reported a predominance of genotype 4 followed by genotype 1a then genotype 1b. (6) Sadeghi et al.,(2016) showed that the genotype 4 was predominant in Saudi Arabia (G4 65%,G123%), Kuwait (G4 43%;G1 28%), Qatar (G4 64%;G1 20%) and Egypt (G4 69%; G1 5%).(2). These results are in contrast to those of Khdeir et al., (Basra 2016) which showed that the genotypes 1a and 1b were the predominant genotypes.(7) The regimen with the highest SVR was (SOF/LDV) followed by SOF/PR and then PR. In the SOF/LDV group, ETR
Comparison of Three Different Treatment Regimens of HCV Infection in 295 Iraqi Patients
Wasan K. Jasim

was 75.6% and SVR was 70.8%, which were low in comparison with ION-1 study (2014) which showed 100% SVR for naïve patients infected with genotype 1(8), ION-3 study (2014) showed an ETR of 100% and SVR of 95%. (9) Gutierrez et al., (2015) showed a SVR 95%.(10) The most responder genotype in SOF/LDV was genotype 4 (74.2%) followed by genotype 1a (68.4%) followed by genotype 1b(58.8%). In ion 2 study the most responder genotype was genotype 1a (95%) followed by genotype 1b (87%). (9) While in ION-3 study the most responder genotype was genotype 1b (98%) then genotype 1a (93%).(9) Zenq et al., (2017) showed SVR of generic SOF/LDV to be 96.9% for G 1b infected patients.(12) Bagaglio et al., in 2015 showed that resistance-associated polymorphisms were prevalently detected in sequences from Europe and in particular in G1b isolates, indicating a different NS5A resistant profile according to geographic origin of subtype. (13) While in the USA, the most resistance genotype was genotype 1a which commonly present to elbasvir/ graziprevir. (14) Francisco in 2014 showed that people with HCV genotype 1 subtype 1a respond more favorably than people with 1b. (15) In case of SOF/PR group; the result of end of treatment was 80% and SVR was 61.1%, while the report of EASL Barcelona 2016 showed that end of treatment (ETR) and sustained viral response rates at week 4 (SVR4) were 99.6% (220/221) and 94.2% (129/137) respectively.(16) Neutrino study (2013) showed that ETR of SOF/PR was 99% and SVR (89%) for G1 (92% of genotype 1a and 82% genotype 1b and 97% for genotype 4) (17). Atomic trial (2013) showed SVR 24 for genotype 1 was 88% and for genotype 4 was 82%(18); Proton study (2013) showed that SVR 24 was 91% for genotype 1(19); Elsharkawy et al., (Egypt 2017) showed a 94% SVR.(20) In the present study SVR is much lower; as in G4, the SVR result was 68.2% follow by genotype 1a (63.6%) and then genotype 1b (50%), similar to the results of Neutrino study in respect to the order of response to therapy.(17) In case of PR group in the present study, the result of ETR was (62.4%) and SVR 48.8%, almost similar to the study of Donato et al., in Italy (2013) where ETR was 59% and SVR 46%(21); as well as the study of Moutaz, etal in 2012 where approximately 62.5% of patients had ETR, and 49.6% had SVR(22) and that of Hassan et al., (Egypt 2015) which showed 51.4% SVR.(23) The most responder genotype to PR was genotype 4 which showed SVR 53.2 %, followed by genotype 1a 46.7% and genotype 1b 40%. The study done by Kamal et al., (2005) (24) in Egypt showed that the ETR was (70%) and SVR 69% for genotype 4 which is higher than the present study (ETR for G4 was 68% and SVR 53.2%). In the present study G1a, ETR was 62.5% and genotype 1b ETR was 58.8% which is similar to the results of Pellicelli et al., (2012) (who showed that G1a ETR and SVR was 65% and 55% respectively; G1b ETR and SVR was 58% and 43% respectively.(25) The PROBE study, a prospective observational multicenter study in Italy, included more than 6000 HCV infected patients, showed that SVR was marginally associated with subtype 1a compared to subtype 1b when treated with PR (OR 1.41; 95% CI 1.0-2.03). (26) While Proton study for genotype 1 showed that SVR 24 was 58%.(27) The International Liver Congress 2016 in Barcelona, Spain, showed high sustained virological response (SVR) after treatment with generic sofosbuvir, ledipasvir, daclatasvir and ribavirin, confirming clinical efficacy equivalent to outcomes seen of branded combination treatments.(16) While such result of generic drugs are not obtained in the present study by use of generic SOF/LDV or generic SOF in SOF+PR which could be due to that sofosbuvir and SOF/LDV used by Iraqi patient were bought by the patients themselves from different sources some of these sources were not under quality control of ministry of health (M.O.H). Presence of resistance of NS5A inhibitors was still possible in our patients as multiple mutations in HCV replicons (genetic units of replication) can cause significant resistance; which was not assessed and can be a cause of low response. Yet in Sofosbuvir, only the single amino acid substitution S282T conferred resistance and decreased the activity of the NS5B inhibitor. This substitution gave a two-fold to 18-fold decrease in susceptibility of the virus to Sofosbuvir. (28)

Conclusions:
Generic SOF/LDV associated with the best response to treatment; which was three times more than PR in achieving SVR and it was statistically significant (Odds ratio = 3.143; 95% CI = (1.591 - 6.208); P=0.001). The most responder genotype in the study group was genotype 4 and the least responder genotype was genotype 1b.

Authors’ contribution:
Wasan Khairet Jasim AL-Saedi :collected samples , wrote the article and made the statistical analysis Nawal Mehdi Firhan AL-Khalidi: help in sample collection, and gave correction advices about article. Ahmed Abass Hussein: reviewed the written article and gave correction advices

References:


15. Franciscus A. Predictors of Treatment response. HCSP 2014(9).


16. EASL. High cure rates using generic hepatitis C drugs bought online: EASL supports lower cost access for Western countries [Internet]. Barcelona: EASL; 2019. Available from: http://i-base.info/hrb/29936


المقارنة بين ثلاثة أنظمة علاجية مختلفة في علاج التهاب الكبد الفيروسي سي ل 295 مريض عراقي

د. وسن خريط جاسم
د. نوال مهدي فرحان الخالدي
د. أحمد عباس حسين

الخلاصة:
خلفية البحث: عندى التهاب الكبد الفيروسي سي، هي مشكلة صحية عالمية في جميع أنحاء العالم، يتم استخدام أنظمة علاجية مختلفة تنتج معدلات استجابة مختلفة تتاثر بالعديد من العوامل.

الأهداف: تقييم فعالية ثلاثة أنظمة علاجية مختلطة في 295 مريض عراقي مصاب بفيروس التهاب الكبد الوبائي سي المزمن.

المرضى والطرق: هي دراسة الم регистра حيث تم تسجيل 295 (133 من الذكور و162 من الإناث) مصابين بعدوى فيروس التهاب الكبد الوبائي سي المزمن في الفترة من 2015 إلى كانون الثاني 2017، من عيادة أمراض الجهاز الهضمي في مستشفى بغداد التعليمي ومستشفى أمراض الجهاز الهضمي والكبد التعليمي. تم الدراسة على مجموعتين أي那就是 المرضى الذين حصلوا للعلاج من طريق ق班子成员 نسبة الفيروسات والتنبؤ الجيني لكل مريض وتم متابعة المرضى الذين قدموا للعلاج عن طريق قومية نسبة الفيروسات في نهاية العلاج وبعد ثلاثة أشهر من انتهاء العلاج.

النتائج: غالبية المرضى المصابين بفيروس التهاب الكبد الوبائي سي المزمن المحققين استجابة فيروسية مستدامه (تعرف بعد تحسن الفيروس بعد 12 إلى 24 أسبوعا من نهاية العلاج) كانوا في مجموعة النظام العلاجي السوفسبوفر/ليديباسفوير بنسبة 70.8% (51 من 72) وبعده السوفسبوفر مع البيكانتيرفرن والريبافرين بنسبة 48.8% (42 من 86) ثلاثة أشهر من انتهاء العلاج.

الاستنتاجات: الدواء الأكثر فعالية هو السوفسبوفر مع البيكانتيرفرن، وهذه الدواء تتميز بعلاج المريض في فترة زمنية قصيرة. وكان النمط الوراثي الأكثر استجابة في المرضى العراقي هو التركيب الوراثي 4 وكن النمط الوراثي الأقل استجابة هو التركيب الوراثي 1.

مفتاح الكلمات: التهاب الكبد الفيروسي سي، سوفسبوفر، أنتيفرن، ريبافرين