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ASSOCIATION BETWEEN VITAMIN D LEVELS AND THE TREATMENT RESPONSES TO SOFOSBUVIR CONTAINING REGIMENS IN CHRONIC HCV EGYPTIAN PATIENTS

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Background *Hepatitis C virus (HCV) is a worldwide epidemic, and it is the highest widespread blood-transmitted infectious viruses which replication occurs in the liver. Nowadays, chronic hepatitis C (CHC) is a very serious health problem. Every year, about 1.5 million newly identified cases of CHC are reported, and 58 million individuals worldwide live with this condition. Also, Egypt records the highest percentage of HCV viral infection around the world. The national program in Egypt achieved a remarkable success in terms of participation rate. From the 62.5 million target population, about 49.6 million individuals (79.4%) performed screening between October 1, 2018, and April 30, 2019.* ***Objective:*** *The aim of the current study was to determine vitamin D levels in both control subjects and HCV patients and the viremia state of HCV patients. Moreover, examine if there is a correlation between vitamin D levels and different parameters in HCV patients before and after treatment.* ***Material and methods:*** *The current study was conducted on two hundred participants; one hundred of them were chronic HCV patients, and the other one hundred were healthy participants as the control group. For every participant, the levels of aspartate aminotransferase (AST), alanine aminotransferase (ALT), total bilirubin, direct bilirubin, ɤ-glutamyl transferase (GGT), albumin, urea, creatinine, triglycerides, total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and glycated hemoglobin A1c (HbA1c) were detected by using the Mindray BA-88A device. Also, the international normalized ratio (INR) was tested using the Yumizen G100 hemostasis analyzer device. The CBC was done by using the Mindray BC-21s device. While the levels of alpha fetoprotein (AFP), vitamin D, tumor necrosis factor alpha (TNF-α), interleukin-6 (IL-6), and thyroid stimulating hormone (TSH) were estimated by using the ELISA strip reader Stat Fax 4700 device. Also, the viral load of HCV was detected using HCV PCR by MX3000 real time PCR equipment.* ***Results:*** *The current study results showed that there was a significant increase regarding to AST, ALT, AFP, GGT, direct bilirubin, total bilirubin, creatinine, urea, HbA1c, WBCs, INR, the inflammatory markers (IL-6 and TNF-α), sun exposure levels, and TSH in HCV patients compared to the control group (P <0.05). While there was a significant decrease regarding to lipid profile (such as triglycerides, total cholesterol, LDL, and HDL), albumin, Hb, and platelets in HCV patients compared to the control group (P <0.05). Also, the vitamin D levels were slightly decreased, but not enough to be a significant change between the HCV patients and the control group (P > 0.05).* ***Conclusion****: Treatment of HCV patients using sofosbuvir-containing regimens improved the liver, renal, lipid profile, and inflammatory parameters.*

Keywords: *HCV, vitamin D, real time PCR, sofosbuvir-containing regimens*

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INTRODUCTION

Nowadays, chronic hepatitis C (CHC) is a very serious healthcare problem. Every year, about 1.5 million newly identified cases of CHC are reported, and 58 million individuals worldwide live with this condition, according to the WHO report for 2021 and approximately 290,000 people who die as a result of CHC each year[1]. Also, Egypt records the highest percentage of HCV viral infection around the world[2]. A successful screening and treatment model was put into practice. DAA HCV treatment was provided for free by the Egyptian National Committee for Control of Viral Hepatitis. A key component of the national program's success was its infrastructure and it had an enormous number of specialized centers. HCV patients received integrated care from these specialized centers and complete care was provided, including examination, assessment, therapy, and follow-up. An experienced multidisciplinary team gave the care. In 2018, there were 64 of these centers around the nation, with less than 50 km separating them all[3]. By 2018, more than two million HCV patients (about 40% of all HCV patients) were treated, and over 90% of those who received treatment as a result of Egypt's well-organised national program had an SVR. It succeeded in achieving great access and readily available for HCV examination, assessment, and therapy. The national program in Egypt achieved a remarkable success in terms of participation rate. From the 62.5 million target population, about 49.6 million individuals (79.4%) performed screening between October 1, 2018, and April 30, 2019[3].

The number of HCV-related fatalities has exceeded the sum of fatalities from 60 other infectious diseases combined, including tuberculosis, pneumococcal disease, and HIV, and the prevalence of HCV infection is rising[4]. Because liver disease is asymptomatic until severe stages, those with HCV infection may present late for care. When seeking therapy for HCV for the first time, European specialists characterized those who presented late for HCV care as having severe fibrosis of the liver[5]. HCV infection is regarded as a systemic disease that influences not just the liver but also additional organs. Furthermore, even though the HCV infection was not diagnosed, extrahepatic manifestations, can be seen in around 75% of patients[6].

In the past few years, direct-acting antiviral (DAA) agents and several promising new drugs have been developed. Sofosbuvir (SOF), which is the generic name of Gilead's Sovaldi, was launched in Egypt in October 2014. HCV-NS5B polymerase is strongly inhibited by SOF, the first DAA medication to be used successfully in Egypt[7]. Sofosbuvir is an antiviral drug acts as a chain terminator of RNA synthesis and has a strong virological response with minimal side effects[8]. As SOF binds to the catalytic site of RNA polymerase and shares structural similarities with its substrate, it restricts HCV replication[9].

By once daily oral dose, is effective against all genotypes, has a high resistance barrier, and is well tolerated[10]. Sofosbuvir is a lipophilic prodrug that undergoes first-pass metabolism in the liver to become its active hydrophilic metabolite[11].

A variety of human illnesses have been related with vitamin D insufficiency, such as infection and inflammatory diseases[12,13], neurological issues, kidney failure, ageing, cancers[14], cardiovascular diseases, reduced muscular strength, type 1 diabetes, systemic lupus erythematosus, rheumatoid arthritis, and ulcerative colitis. Demonstrating its significance for human health. This is because vitamin D is essential for regulating collagen metabolism, skeletal muscle size and function, and inflammatory processes[15]. Vitamin D is a family of compounds consisting of 9-10 steroids in the following five forms: Ergosterol or Ergocalciferol (D2) (25(OH)D2 ergocalciferol), Cholecalciferol (D3) (25(OH)D3; cholecalciferol), 22,23 dihydroergocalciferol (D4), Sitosterol or 24-ethylcholecalciferol (D5), and Stigmasterol (D6)[16].

Vitamin-D is an important immune regulator and prevent the immunosuppressive effect of PD-L1[17], the latter is responsible for T cell activation, proliferation, and cytotoxic secretion[18]. Toll-like receptors 2 and 4 (TLR2 and TLR4), play a considerable role in the host defense against microorganism[19]. Both TLR2 and TLR4 on monocytes have been shown to be downregulated by the immunomodulatory effect of vitamin D[20]. A significant relationship

between key regulators of immune homeostasis[21], TGF-β and vitamin D deficiency get established[22].

It is known that vitamin D deficiency is related to the risk of cancer, autoimmune diseases, infectious diseases, and cardiovascular diseases[23]. Numerous human diseases, including those caused by alcoholism, viral hepatitis, and NAFLD, are all clearly linked to low vitamin D serum levels. Additionally, these circumstances speed up the development of chronic liver disorders and have a clearly negative effect on clinical results[24]. Numerous chronic liver diseases, including chronic hepatitis C, primary biliary cholangitis, autoimmune hepatitis (HIV), non-alcoholic fatty liver disease (NAFLD), advanced cirrhosis, and hepatocellular carcinoma, have been linked to vitamin D deficiency[25]. Additionally, higher mortality, infections, portal hypertension outcomes, and liver cirrhosis have all been linked to vitamin D insufficiency due to the liver's role in the pleiotropic process and vitamin D metabolism[26].

Liver diseases hinder the formation of vitamin D's active metabolites, which leads in improper calcium and bone metabolism[27]. There are several studies which have shown that vitamin D deficiency is related to HCV infection[28]**,** Vitamin D has anti-inflammatory characteristics, thus its lack in HCV-positive patients worsens chronic inflammation. In patients with chronic liver disease, a lack of vitamin D patients will result in the advancement of hepatic fibrosis. Hepatocytes and hepatic stellate cells are activated by vitamin D receptors, which inhibit stellate cell proliferation. Haematopoietic stem cell (HSC) activation is inhibited by vitamin D receptor (VDR) ligands, which also prevent liver fibrosis[27].

Also, there are studies that have recorded that taking vitamin D activates the innate and adaptive immune responses to HCV viral infection through inhibition of the replication of the virus[29].

Vitamin D deficiency reduces treatment efficiency and is related to severe fibrosis in chronic HCV patients. As it helps in suppression of pro-inflammatory cytokines, increasing production of anti-inflammatory cytokines, and helping to improve the T-cell hyperreactivity.[8] And vitamin D suppresses viral replication, reverses hepatic fibrosis, and improves the virological response in chronic HCV patients whether they have cirrhosis or not.[30]

By using oxidative stress pathways, vitamin D may suppress the replication of the HCV virus. Vitamins D3, D2, and 1,25(OH)2D3 work together to inhibit HCV replication when combined with IFN-alfa in cell culture. Additionally, vitamins D3 and D2 can help to clear HCV in vivo by promoting the gene expression of CCL20 (macrophage inflammatory protein-3 alpha)[31]. And thus, the current study's aim was to detect the level of vitamin D among HCV patients before and after treatment.

MATERIALS AND METHODS

I) Sample collection

The current study included two hundred participants: 100 chronic HCV patients (58 males and 42 females) whose ages ranged between (22-67) years old, and 100 healthy subjects as the control group (54 males and 46 females) whose ages ranged between (30-67) years old. Also, after 12 weeks of treatment HCV patients with sofosbuvir-containing regimens, biochemical tests were repeated. This study was conducted during the period between July 2022 and May 2023.

Inclusion criteria

HCV patients with a mild and moderate viremia status confirmed by PCR (viral load).

Exclusion criteria

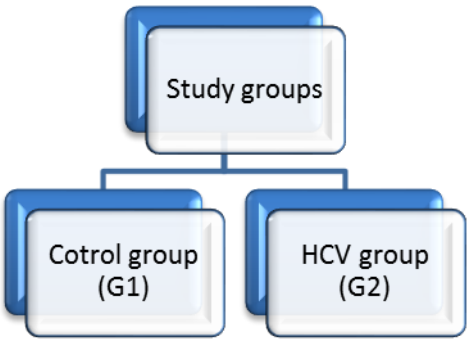
Hepatitis B virus (HBV), HIV virus, pregnancy, and alcohol consumption.

Ethics and informed consent

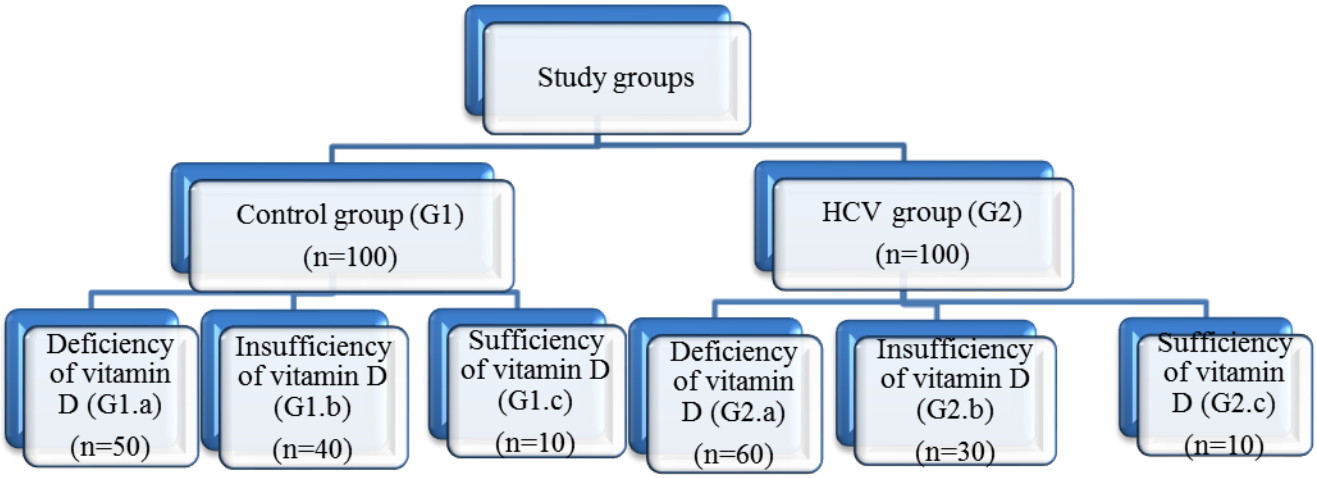
The ethical approval was taken from the National Hepatology and Tropical Medicine Research Institute (NHTMRI) -Cairo with serial number: 18-2022.

II) The current study was classified into the following categories:

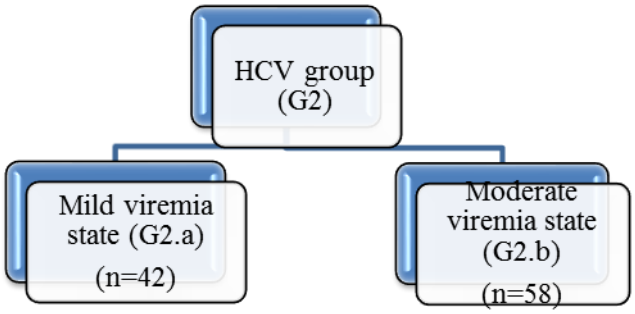
A)


Study groups
Cotrol group (G1)
HCV group (G2)

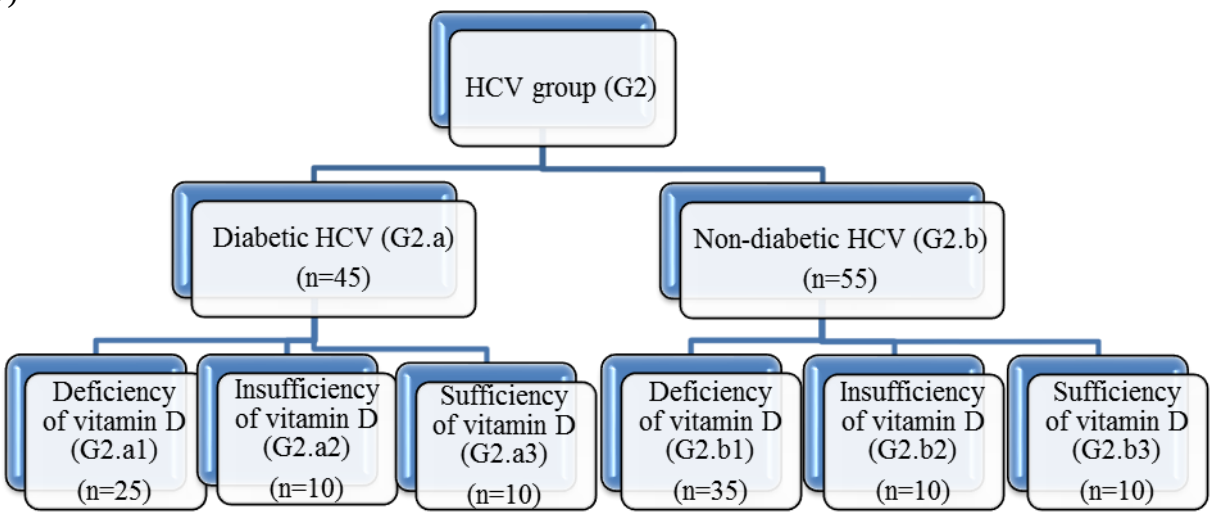
B


Study groups
Control group (G1) (n=100)
HCV group (G2) (n=100)
Deficiency of vitamin D (G1.a) (n=50)
Insufficiency of vitamin D (G1.b) (n=40)
Sufficiency of vitamin D (G1.c) (n=10)
Deficiency of vitamin D (G2.a) (n=60)
Insufficiency of vitamin D (G2.b) (n=30)
Sufficiency of vitamin D (G2.c) (n=10)

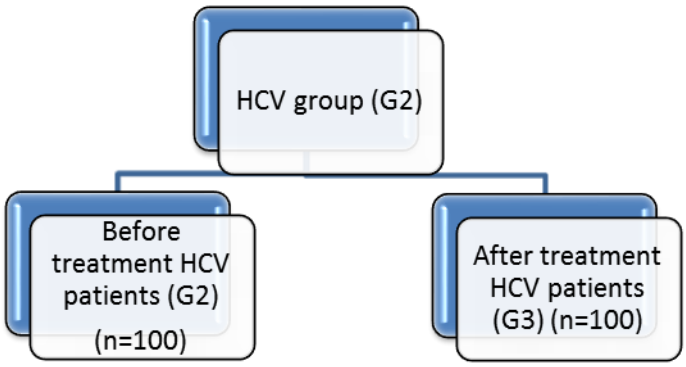
C)


HCV group (G2)
Mild viremia state (G2.a) (n=42)
Moderate viremia state (G2.b) (n=58)

D)


HCV group (G2)
Diabetic HCV (G2.a) (n=45)
Non-diabetic HCV (G2.b) (n=55)
Deficiency of vitamin D (G2.a1) (n=25)
Insufficiency of vitamin D (G2.a2) (n=10)
Sufficiency of vitamin D (G2.a3) (n=10)
Deficiency of vitamin D (G2.b1) (n=35)
Insufficiency of vitamin D (G2.b2) (n=10)
Sufficiency of vitamin D (G2.b3) (n=10)

E)



Biochemical analysis

Liver function tests

The Mindray BA-88A device was used to measure the levels of alanine aminotransferase (N.S. BIO-TEC)[32], aspartate aminotransferase (N.S. BIO-TEC)[33], ɤ-glutamyl transferase (Accurex biomedical, GT-2009-06-001, Mumbai)[34], total bilirubin (Biomed, BIL099250, Egypt)[35], direct bilirubin (Biomed, BIL099250, Egypt)[35], albumin (Biomed, ALB 100250, Egypt)[36]. In addition to using the ELISA strip reader Stat Fax 4700 device to detect alpha fetoprotein levels (Panomics, BC1009)[37].

Renal function tests

The levels of creatinine (N.S.BIO-TEC)[38], and urea (Biomed, URE118200, Egypt)[39] were estimated by the Mindray BA-88A device.

Lipid profile

The Mindray BA-88A device was used to detect the levels of total cholesterol (Dialab, D00123, Austria)[40], high-density lipoprotein (Biomed, HDL114100, Egypt)[41], low-density lipoprotein (Biomed, HDL114100, Egypt) [41], and triglyceride (Dialab, D81911, Austria)[42].

Hematology tests

The levels of hemoglobin, platelets, and white blood cells were detected using the Mindray BC-21s device. While glycated hemoglobin A1c was measured using the Mindray BA-88A device (Biomed, Egypt) [43]. Also, an international normalized ratio was detected using the Yumizen G100 hemostasis analyzer device (Horiba medical ,1300036417, France)[44].

Inflammatory markers

The ELISA strip reader Stat Fax 4700 device was used to measure interleukin-6 (Abbexa, abx050124, UK)[45], and tumor necrosis factor alpha (Eagle bioscience, K 9610, Germany)[46].

Also, the levels of vitamin D levels (PerkinElmer, Cabot Blvd., Suite, Hayward, CA)[47] and thyroid stimulating hormone (BIOS, 10303, South San Francisco)[48] were tested by using the ELISA strip reader Stat Fax 4700 device.

Statistical analysis

The data were analyzed using SPSS statistics version 19. A T-test and one-way ANOVA using the Scheffe test were used to find the significant difference between groups and subgroups at $P< 0.05$. Person-to-person correlation was used to test the presence of a significant relation between two variables.

RESULTS AND DISCUSSION

Vitamin D is regarded as inactive biologically, and its hydroxylation into 25-hydroxyvitamin D [25(OH)D] occurs in the liver[49]. So, liver diseases prevent the production of the active metabolites of vitamin D, leading to incorrect metabolism of calcium and bone[50].

Vitamins D2 and D3 are both biologically inactive, and there are two processes involved in the hydroxylation reaction that activates vitamin D. The liver's first hydroxylation at the 25th carbon atom is considered one of the causes linking chronic liver disease to vitamin D deficiency. Then, in the kidney, the first carbon of calcidiol undergoes the next

hydroxylation. This results in the production of 1,25(OH)2D3, or calcitriol, the fully activated form of vitamin D[51].

The current study's findings that HCV patients had significantly increase levels of ALT, AST, and γ-GT when compared to the control group, and it was supported by pervious study by Giuffrè, M et al. 2020[52], who suggested that there was a link between these enzymes and liver-related disease and mortality.

Table1: Results of biochemical parameters in control group and HCV patients.

Groups Parameters	Control group	HCV group
Vitamin D (ng/dl)	19.8 ± 0.7	18.02 ± 0.6
Sun exposure (min/day)	113.7 ± 2.2	127.7 ±2.5[a]
ALT (U/L)	30.13 ± 0.56	47 ± 1.5[a]
AST (U/L)	30.3 ± 0.4	50.1 ± 1.4[a]
AFP (ng/dl)	5.6 ± 0.15	15 ± 0.6[a]
Total Bilirubin (mg/dl)	0.77 ± 0.019	0.96 ± 0.031[a]
Direct Bilirubin (mg/dl)	0.15 ± 0.006	0.21 ± 0.01[a]
Albumin (g/dl)	4.54 ± 0.04	3.15 ± 0.03[a]
GGT (IU/L)	35.2 ± 0.97	55 ± 1.8[a]
Creatinine (mg/dl)	0.94 ± 0.01	1.03 ± 0.02[a]
Urea (mg/dl)	29.5 ± 0.7	34.7 ± 0.9[a]
Total cholesterol (mg/dl)	195.1 ± 2.5	160.5 ± 2.1[a]
LDL (mg/dl	126.7 ± 2.6	111.9 ± 1.17[a]
HDL (mg/dl)	42.7 ± 0.7	37.4 ± 0.8[a]
Triglyceride (mg/dl)	189.1 ± 3.7	157.4 ± 2.7[a]
Hb (g/dl)	13 ± 0.1	10.9 ± 0.13[a]
WBCs (cell/ mm^3)	6636.3 ± 209.4	6818.8 ± 164.9[a]
Platelets (cell/ mm^3)	227270 ± 5468.4	120480±1231[a]
INR	1.06 ± 0.008	1.1 ± 0.012[a]
HbA1c (%)	3.1 ± 0.09	5.9 ± 0.15[a]
TSH (μIU/ml)	2.2 ± 0.05	3.6 ± 0.09[a]
Interleukin-6 (IL-6) (pg/ml)	21 ± 0.5	28.4 ± 0.9[a]
Tumor necrosis factor (TNF) (pg/ml)	16.4 ± 0.6	19.9 ± 0.2[a]

The data was shown as mean ± SE. Lowercase letters indicated a statistically significant ($P<0.05$) difference.
[a]($P<0.05$) with respect to the control group.

According to the current study, HCV patients' AFP levels were significantly increase than those of the control group. As in patients with viral hepatitis, AFP acts as a dependent marker of liver regeneration following hepatocyte damage and as a marker for HBV and HCV patients. and similar results were detected by Yang, J.D et al. 2017[53] who suggested that there was a correlation between elevation of AFP and an increase of ALT and a decrease in albumin and platelet levels.

The current study showed that, compared to the control group, HCV patients' albumin levels significantly decreased. as albumin synthesis was lowered as a result of chronic liver illness, and it was supported by Ramadan MA et al. 2021[54] who demonstrated that after treatment with DAAs there was an increase in the level of albumin.

According to the current study, HCV patients' bilirubin levels were significantly increase than those of the control group, and it was supported by previous research by Ramadan MA et al. 2021[54].

The current study's findings that HCV patients had significantly increase levels of urea compared to the control group and it was supported by a previous study by Hassan AH.2023[55] who suggested that urea is considered the main byproduct of the protein catabolism process and is produced in the liver, put into the bloodstream, and then eliminated by the kidneys. Also, it is considered a helpful biomarker of hepatic and renal integrity.

According to the current study, HCV patients' creatinine levels were significantly increase than those of the control group, and it was supported by a previous study by Hassan AH.2023[55].

Lipid metabolism is primarily regulated by the liver, The hepatitis C virus has been linked to the development of chronic liver disease and reduced lipid profiles. HCV has been linked in numerous studies to abnormal lipid profiles, which raise the risk of liver steatosis, progressive fibrosis, and dyslipidemia[56].

According to the current study, HCV patients' lipid profile levels were significantly decrease than those of the control group, and it was agreed by Gad Allah SH. et al 2022[56] who suggested that lipid metabolism is primarily regulated by the liver, and the hepatitis C virus has been linked to both the onset of chronic liver disease and lowered lipid profiles.

The current study's findings that HCV patients had significantly decrease levels of platelet and hemoglobin compared to the control group and it was agreed by previous research by Ramadan MA et al.2021[54] who demonstrated that HCV is related to a decrease in the value of platelets and hemoglobin and by Seko Y. et al. 2020[57], who showed that the significant contributor of thrombocytopenia in HCV patients was platelet-associated immunoglobulin G (PA-IgG).

According to the current study, HCV patients' IL-6 levels were significantly increase than those of the control group, and it was supported by Noh, I.C. et al. 2022[58], who demonstrated that A pleiotropic cytokine having pro- and anti-inflammatory properties, IL-6 is quickly generated by different cells in response to tissue damage and infection, such as vascular endothelial cells, mast cells, fibroblasts, macrophages, B cells, and T cells and by Sghaier I et al. 2017[59] who said that viral-induced inflammation increased blood IL-6 levels.

The current study's findings that HCV patients had significantly increase in TNF-α levels compared to the control group, and it was supported by Noh, I.C. et al. 2022 [58], who demonstrated an elevation in the TNF-α and IL-6 levels while decreasing protein or albumin levels, indicating extensive liver damage.

The current study showed that there was a significant increase in the levels of TSH in HCV patients compared to the control group, and the results were supported by a previous study carried out by El-Feki MA et al.2016[60] who demonstrated that both thyroid autoimmunity and hypothyroidism were common in HCV patients.

The current study showed that there were slightly decreased in the vitamin D levels, but not enough to be a significant change between the HCV patients and the control group and that was confirmed by previous studies carried out by Kefeli A, and Demir AK, 2020[61] and Basile U. et al. 2019 [62], who believed that vitamin D deficiency was a serious global public health issue, whereas Gabr SA. et al. 2016[63] proposed that vitamin D levels decreased in individuals with HCV genotype 4.

In the treated HCV group, the decrease in liver function tests was supported by previous study carried out by Doğan M et al 2020[64]. Also, the increase in albumin levels and the decrease in INR levels were confirmed by Ibrahim, E.et al 2021[65].

In the treated HCV group, the decrease in creatinine was supported by previous study by Dala AG et al 2020[66]. While the decrease in urea was confirmed by previous study carried out by Shaaban MA et al 2019[67].

Within the HCV treatment population, the increase in lipid profile was agreed by previous study carried out by El-Lehleh, A. M et al. 2019[68] who showed that the reversal of the effects of HCV replication on hepatic lipid metabolism is believed to be the cause of this effect.

In the current study before starting the treatment for hepatitis C patients, the percentage of diabetic patients was 45% of the total number of hepatitis C patients, and they had type 2 diabetes, while the percentage of diabetic patients became 2% after treatment with reduction of 95.5%.

Among the HCV-treated group, the decrease in HbA1c was confirmed by another study carried out by Abdo M et al. 2021[69]**,** who suggested that effective HCV removal after treatment resulted in an improvement in insulin resistance and glycemic state.

In the treated HCV group, the increase in Hb and platelets was supported by Dala AG et al .2020[66].

Among the treated HCV group, the decrease in interleukin-6 was supported by another study carried out by Saafan AE et al. 2020[70] who suggested that sofosbuvir therapy regimens eradicate the virus and restore the immune system.

Within the treated HCV group, the decrease in TNF-α was supported by a previous study by Salem H et al.2022[71] who suggested that since TNF-α is implicated in hepatocyte death and liver inflammation, inhibiting it could be beneficial for the eventual clearance of HCV. Additionally, up-regulating TNF-α pathways was believed to impact therapy non-response.

Among the HCV-treated group, the TSH levels were decreased compared to the before treatment levels while Dala AG et al .2020[66] found that there was no significant change in TSH levels after treatment.

In the current study vitamin D supplementation was taken by the treated HCV group, to enhance the immune system and the response to the treatment. The mean ±SE of vitamin D in the HCV group before treatment and the HCV group after treatment was 18.02± 0.6 and 29± 0.73, respectively so there was a significant increase among HCV groups.

Also, there were a lot of studies that have recorded that taking vitamin D activates the innate and adaptive immune responses to HCV viral infection through inhibition of the replication of the virus[29].

Vitamin D deficiency reduces treatment efficiency and is related to severe fibrosis in chronic HCV patients; also, vitamin D suppresses viral replication, reverses hepatic fibrosis, and improves the virological response in chronic HCV patients, whether they have cirrhosis or not. [(8)]

Conclusion

In the current study the results showed no effect of vitamin D in HCV patients as many healthy subjects also suffered from its deficiency. Also, there is no effect of HCV viremia state and diabetes mellitus among HCV patients. Treatment of HCV patients using sofosbuvir-containing regimens improved the liver, renal, lipid profile, and inflammatory parameters.

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الارتباط بين مستويات فيتامين د والاستجابات العلاجية للأنظمة التي تحتوي على سوفوسبوفير في المرضى المصريين المصابين بفيروس التهاب الكبد سي المزمن

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يعد فيروس التهاب الكبد الوبائي سي وباء عالمي، وهو أكثر الفيروسات المعدية المنقولة عن طريق الدم انتشارا والتي يحدث تكاثرها في الكبد. وفي الوقت الحاضر، يمثل التهاب الكبد الوبائي سي المزمن مشكلة صحية بالغة الخطورة. ففي كل عام، يتم الإبلاغ عن حوالي ١.٥ مليون حالة جديدة تم تشخيصها حديثًا من المرض، ويعيش ٥٨ مليون شخص حول العالم بهذه الحالة. كما تسجل مصر أعلى نسبة إصابة بفيروس التهاب الكبد الوبائي سي على مستوى العالم، وحقق البرنامج الوطني في مصر نجاحاً ملحوظاً من حيث نسبة المشاركة. من بين عدد السكان المستهدف البالغ ٦٢.٥ مليون فرد، أجرى حوالي ٤٩.٦ مليون فرد بنسبة (٧٩.٤%) الفحص في الفترة ما بين ١ أكتوبر عام ٢٠١٨ و٣٠ أبريل عام ٢٠١٩. تهدف الدراسة الحالية الى:

١) قياس معدلات فيتامين د في كل من مجموعه مرضي التهاب الكبد الوبائي سي ومجموعة المشاركين الأصحاء.

٢) تحديد حالة فيرس التهاب الكبد الوبائي سي في الدم.

٣) فحص ما إذا كانت هناك علاقة بين فيتامين د والمعايير الأخرى في مرضي التهاب الكبد الوبائي سي قبل وبعد العلاج.

أجريت الدراسة الحالية على مائتي مشارك؛ مائة منهم كانوا مصابين بفيروس التهاب الكبد الوبائي سي المزمن، والمئة الآخرين كانوا مشاركين أصحاء كمجموعة مراقبة.

وبالنسبة لكل مشارك تم الكشف عن مستويات انزيم ناقلة أمين الأسبارتات، انزيم ناقلة أمين الألانين، البيليروبين الكلي، البيليروبين المباشر، ناقلة جاما جلوتاميل، الألبومين، اليوريا، الكرياتينين، الدهون الثلاثية، الكوليسترول الكلي، البروتين الدهني مرتفع الكثافة، البروتين الدهني منخفض الكثافة، والهيموجلوبين السكري.

كما تم أيضا اختبار لنسبة المعيارية الدولية، تعداد الدم الكامل، ألفا فيتوبروتين، عامل نخر الورم ألفا، الإنترلوكين-٦، فيتامين د، والهرمون المحفز للغدة الدرقية، والحمل الفيروسي لفيروس التهاب الكبد الوبائي سي.

وأظهرت نتائج الدراسة الحالية ان هناك ارتفاع ملحوظ في مستويات انزيم ناقلة أمين الأسبارتات، انزيم ناقلة أمين الألانين، البيليروبين الكلي، البيليروبين المباشر، ناقلة جاما جلوتاميل، ألفا فيتوبروتين، الكرياتينين، اليوريا، الهيموجلوبين السكرى، علامات الالتهاب (عامل نخر الورم ألفا، الإنترلوكين-٦)، خلايا الدم البيضاء، النسبة المعيارية الدولية، مستويات التعرض للشمس، والهرمون المحفز للغدة الدرقية في مرضى التهاب الكبد الوبائي سي مقارنة بمجموعة المشتركين الاصحاء. في حين كان هناك انخفاض ملحوظ فيما يتعلق بملف الدهون (مثل الدهون الثلاثية والكوليسترول الكلي والبروتين الدهني منخفض الكثافة والبروتين الدهني مرتفع الكثافة) والألبومين والهيموغلوبين والصفائح الدموية في مرضى التهاب الكبد الوبائي سي مقارنة بمجموعه المشتركين الاصحاء. كما انخفضت مستويات فيتامين د بشكل طفيف، ولكن ليس بدرجة كافية لإحداث تغيير كبير بين مرضى التهاب الكبد الوبائي سي ومجموعة المشتركين الاصحاء. الاستنتاج: أدى علاج مرضى التهاب الكبد الفيروسي سي باستخدام الأنظمة العلاجية المحتوية على سوفوسبوفير إلى تحسن في وظائف الكبد والكلى ومستوى الدهون ومعايير الالتهاب.