Biochemical markers and physiological functions in Egyptian patients with hepatitis C virus

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ABSTRACT

Infection with the Hepatitis C virus (HCV) is a severe public health concern worldwide. HCV is a prevalent cause of liver fibrosis, which can progress to cirrhosis or hepatocellular cancer. To assess the research objective, we evaluated parameters including liver functions, liver tumor markers, hematological, renal functions, lipid profile, and blood sugar in two groups: the healthy control group with the group of hepatitis C virus patients. In the presented study, albumin (ALB), alkaline phosphatase (ALK PH), and Bilirubin total (BIL T) were compared, and there were no remarkable variations between the control and patient groups. On the other hand, γ-glutamyl transaminase (GGT), aspartate transaminase (AST), alanine transaminase (ALT), and bilirubin direct (Bill D) showed noticeable differences between the two groups. Regarding the lipid profile, including cholesterol (CHO), triglyceride (TG), low-density lipoprotein (LDL), and high-density lipoprotein (HDL), There were no statistically significant variations between the control and patient communities. Therefore, data analysis for HCV biochemical markers and physiological activities was critical for understanding HCV's epidemiology and treatment methods in Egyptian chronic patients.

Keywords: Hepatitis C virus; Biochemical markers; Physiological function; Egyptian patients.

INTRODUCTION:

Almost 170 million individuals worldwide are infected with HCV, which is the main reason for liver fibrosis, cirrhosis, and hepatocellular carcinoma. Currently, most treatment pharmaceuticals are direct-acting medicines such as protease, polymerase, and polymerase accessory protein inhibitors, with interferon (IFN) and ribavirin combination therapy even now playing significant participation, particularly in certain poor nations. Even though HCV is a hepatotropic virus, it has also been discovered in tissues exterior to the liver. Various extrahepatic symptoms have been linked to HCV infection (Yinping *et al*., 2019). HCV is transmitted via parenteral routes. Contaminated blood products constitute a significant source of infection, which is why the phrase "post-transfusion hepatitis" was created (Dietz *et al*., 2022).

Eradication of HCV with direct-acting antivirals (DAA) is associated with improved outcomes at all stages of liver disease, and the development of DAA against HCV has improved hepatitis C treatment (Maria Giovanna *et al*., 2021). Changes in lipid levels have been linked to HCV infection. When compared to demographically similar HCV uninfected controls, people with HCV had considerably reduced Triglycerides, total cholesterol (TC), and low-density lipoprotein (LDL) triglyceride (TG) values. Others have demonstrated alterations in lipid profiles following HCV therapy (Adeel *et al*., 2015). Although hepatitis C virus infection is frequently associated with a disruption in glucose metabolism, there have been several variances in HCV clearance, followed by variations in blood glucose and insulin resistance. Examine the effect of HCV and antiviral therapies on blood glucose levels and other glucose metabolic markers. Insulin resistance (IR) was assumed to be caused by HCV infection. (Yinping *et al*., 2019).

Chronic hepatitis C virus infection can impair kidney function by causing systemic inflammation or raising the hazard of chronic kidney disease risks factors such as metabolic syndrome, diabetes, mixed cryoglobulinemia, cardiovascular disease, membranoproliferative glomerulonephritis, and lymphoproliferative disorders. (Cheng *et al*., 2021).

The only currently licensed NI treatment, sofosbuvir, is a phosphoramidite prodrug that is degraded intracellularly and then transformed into the active triphosphate structure, a uridine analog with a 2fluoro-C-methyl motif. Because of steric conflict with incoming nucleotide substrate, this motif is regarded to be the cause of chain termination. Even though first certified by PEG-IFN and ribavirin, sofosbuvir is extremely potent when taken with other direct-acting antiviral groups and dual or triple direct-acting antiviral

combinations in an interferon-free setting. Although sofosbuvir is typically approved and has a favorable safety profile, liver toxicity incidences in people with decompensated cirrhosis have been reported. (Dietz *et al*., 2022).

The current study aims to evaluate the biochemical and physiological activities of the liver, kidney, lipid profile, blood sugar, and tumor marker in HCV patients treated with Sofosbuvir and a healthy control group.

Patient and methodologies

Thirty blood samples were collected (15 patients with hepatitis C virus and 15 healthy people with clinical signs and according to the doctor's diagnosis from the medical laboratory unit in Military Mostafa Kamel Hospital in Alexandria from (1/1/2017 to 1/1/2018). The samples were separated into two tubes for each patient, then frozen and stored immediately (-20 ° C) and -80°C. All the chemical kits were used according to the manufacturer's instructions.

There were 15 people with a confirmed chronic hepatitis C virus diagnosis. The control group consisted of 15 healthy people, equivalent to the experimental group regarding core clinical features. SPAIN REACT Company used all the chemical reagents.

Liver function

Aspartate transaminase (AST) or Glutamate oxaloacetate transaminase (GOT): R1a. Buffer /substrate:

Tris buffer: 80 mmol/l, pH 7.5

L-aspartate: 240mmol/l

R1b. Enzyme/Coenzyme/alpha-oxoglutrate:

alpha-oxoglutrate 12mmol/l

MDH 420 U/l

LD 600 U/l

NADH 0.18 mmol/l

α-oxoglutarate reacts with L-aspartate in the presence of AST to form L-glutamate plus oxaloacetate. The indicator reaction utilizes oxaloacetate for a kinetic determination of NADH consumption.

$$\text{Alpha} - \text{oxoglutarate} + \text{L} - \text{aspartate} \xrightarrow{AST} \text{L} - \text{glutamate} + \text{oxaloacetate}$$

$$\text{oxaloacetate} + \text{NADH} + \text{H}^+ \xrightarrow{MDH} \text{L} - \text{malate} + \text{NAD}^+$$

Kinetic method: R1a. Buffer /substrate with volume 400 microLiter + R2 (AST coenzyme) 100microLiter then (working reagent) incubate at 37 degrees for 5min then add 50 microliters from a serum sample, mix, incubate for 1.0 minutes, and start stopwatch simultaneously. Reread the absorbance after precisely 1, 2, and 3 minutes.

Alanine aminotransferase (ALT) or Glutamate pyruvate transaminase (GPT):

R1. Tris buffer: 100 mmol/l, pH 7.8

L-Alanine: 500 mmol/l

LDH: 1200 U/l

R2. 2-oxoglutrate: 15mmol/l

NADH: 0.18 mmol/l

$$L - Alanine + 2 - Oxoglutarate \xrightarrow{ALT} Pyruvate + L - Glutamate$$

$$Pyruvate + NADH + H^+ \xrightarrow{LDH} L - Lactate + NAD^+$$

The coenzyme NADH's oxidation rate is proportional to the ALT activity in the specimen. It is determined by measuring the decrease in absorbance at 334 / 340 / 365 nm correspondingly. Endogenous specimen pyruvate is rapidly and completely reduced by LDH during the initial incubation period so that it does not interfere with the assay.

Kinetic method: R1. Buffer /substrate: with volume 400 microLiter + R2 (ALT coenzyme) 100microLiter then (working reagent) incubate at 37 degrees for 5min then add 50 microliters from a serum sample, mix, incubate for 1.0 minutes, and start stopwatch simultaneously. Read again after exactly 1, 2, and 3 minutes.

Albumin (Alb): Colorimetric determination of serum albumin.

R1: Albumin standard 4.0 g/dl

R2: Succinate buffer 75.0 mmol/l, pH 4.2

Bromcresol green 0.26 mmol/l

Taking 2.5 ml albumin reagent + 10 microliter of a serum sample, incubate the mix for 10 min at room temperature, then read absorbance.

$$Albumin + BCG \xrightarrow{pH4.2} Albumin - BCG\ complex$$

The intensity of the blue-green color is directly proportional to the albumin concentration in the specimen. It is determined

by measuring the increase in absorbance at 580 - 630 nm.

Bilirubin total (Bil T):

R1: Sulfanilic acid 30 mmol/L

Hydrochloric acid (HCl) 50 mmol/L

Dimethylsulphoxide (DMSO) 7 mol/L

R2: Sodium nitrite 29 mmol/L

Preparation: Pipette 1,5 mL of R2 into R1 content. Mix, avoiding foam forming, and it will be ready to use (WR). Do not use the reagent before 30 min. after the reagent preparation.

Principle of the method: Bilirubin is converted to colored azobilirubin by diazotized sulfanilic acid and measured photometrically. Of the two fractions present in serum, bilirubin-glucuronide, and free bilirubin loosely bound to albumin, only the former reacts directly in aqueous solution (bilirubin direct), while free bilirubin requires solubilization with dimethylsulphoxide (DMSO) to react (bilirubin indirect). In the determination of indirect bilirubin, the direct is also determined; the results correspond to total bilirubin. The intensity of the color formed is proportional to the bilirubin concentration in the sample.

• γ-glutamyl transferase (GGT): For the quantitative kinetic determination of serum gamma-glutamyl transferase (GGT) activity.

Clinical Significance GGT measurements are used to diagnose and treat liver diseases such as alcoholic cirrhosis and primary and secondary tumors. Elevated GGT levels appear earlier and are more pronounced than those of other liver enzymes in cases of obstructive jaundice and metastatic neoplasms.

R1: Tris buffer (pH 8.1 ±0.1) 89 mmol/L

Glycylglycine 126 mmol/L

R2: GLUPA-C 3.3 mmol/L

Reagent Composition In addition to a stabilizer, the combined R1 and R2 reagent contains Sodium Azide 0.095% as a preservative.

Principle :

$$L-y-Glutamyl-3-carboxy-4-nitroanilide + Glycylglycine \xrightarrow{GGT}$$

$$L-y-glutamylglycylglycine + 5-amino-2-nitrobenzoate$$

Kinetic method: R1(substrate)400 micro + R2 100 micro, incubate for 5 min at 37 c, add 5 micro samples, and read absorbance.

GGT in the sample catalyzes the transfer of the glutamyl group from GLUPAC to glycylglycine according to the above reaction. The amount of 5-amino2-nitrobenzoate formed is proportional to GGT activity and may be measured kinetically at 405nm.

Alkaline phosphatase: Kinetic determination of serum alkaline phosphatase (ALK Ph)

R1: Diethanolamine buffer pH 9.8, 1.0mol/l

Magnesium ions 0.6 mol/l

R2: p-Nitrophenyl phosphate 10 mmol/l

$$p-nitrophenylphosphate + H2O \xrightarrow[Mg++]{ALP} p-nitrophenol + Phosphate$$

Alkaline phosphatase (ALP) hydrolyzes the colorless pnitrophenyl phosphate to p-nitrophenol and phosphate in the presence of magnesium ions. The product of enzyme hydrolysis, p-nitrophenol, has a yellow color at the pH of the reaction. The rate of p-nitrophenol formation is directly proportional to the catalytic ALP activity. It is determined by measuring the increase in absorbance at 405 nm.R1 500 micro + R2 500 micro + 20 micro serum /plasma, then incubate the mix for 1 min, then read.

Tumor marker: AFP (alpha-fetoprotein): Using: ELISA FOR AFP, sample size: 25 Microliter, incubation time: 1.5 hours, and dynamic range: 5-200 ng/ml

Hematology withdrawing blood sample on anticoagulant additive (potassium Ethylene Di-amine Tetra Acetic Acid) (K.EDTA) anticoagulant and mix well; finally put it into CBC device, will count hemoglobin, RBC, WBC with differential and platelets.

Lipid profile

Cholesterol (CHO): Enzymatic Colorimetric Determination of Serum Cholesterol.

R1. Cholesterol standard 200 mg/dl

R2. Pipes buffer,

pH 6.9 90 mmol/l

Phenol 26mmol/l

Cholesterol oxidase 500U/l

Cholesterol esterase 500U/l

Peroxidase 1250U/l

4-Aminoantipyrine 0.4 mmol/l

1ml cholesterol reagent + 10 microliter serum sample, then incubate for 10 min at 37c and read absorbance.

$$Cholesterol\ esters + H2O \xrightarrow{CHOD} Cholesterol + fatty\ acids$$

$$Cholesterol + O2 \xrightarrow{CE} Cholestt - 4 - ene - 3 - one + H2O2$$

$$2\ H2O2 + 4 - AAP + Phenol \xrightarrow{POD} Quinoneimine\ dye + 4H2O$$

The intensity of the color produced is directly proportional to cholesterol concentration. It is determined by measuring the increase in absorbance at 500 – 550 nm.

Triglycerides: Colorimetric method 1ml triglycerides reagent + 10 microliter serum sample, then incubate for 10 min at 37 c and read absorbance.

R1. Triglycerides standard 200 mg/dl

Pipes buffer50mmol/l

R2.,

8 p-Chlorophenol 2.0 mmol/l

Lipoprotein lipase 1500 U/l

Glycerolkinase 800 U/l

Glycerol phosphate oxidase 4000 U/l

Peroxidase 440U/l

4-Aminoantipyrine 0.4 mmol/l

ATP 0.3 mmol/l

Mg++ 40 mmol/l

Sodium Cholate 0.2 mmol/l

$$Triglycerides \xrightarrow{LPL} Glycerol + fatty\ acids$$

$$Glycerol + ATP \xrightarrow{GK} Glycerol - 3 - phosphate + ADP$$

$$Glycerol - 3 - phosphate + O2 \xrightarrow{GPO} Dihydroxyacetone\ phosphate^{+}$$

$$2\ H2O2 + 4 - AAP + 4 - Chlorophenol \xrightarrow{POD} Quinoneiminedye + 4\ H2O$$

High-Density Lipoprotein (HDL): HDL-precipitating reagent is intended for the in vitro quantitative separation of High-Density Lipoproteins (HDL) in serum on manual systems.

The reference procedure for separating the different lipoprotein density classes is density-adjusted ultra-centrifugation, which is not practical for routine laboratory use. Widely used alternatives are based on polyanion precipitation techniques. Polyanions such as heparin, dextran sulfate, and phosphotungstate bind to positively charged Apoprotein B-containing lipoproteins cross-linked by a divalent cation (manganese, magnesium, or calcium) to form a precipitate. The NS Biotec HDL-precipitating method utilizes the well-established precipitating properties of phosphotungstic acid to precipitate non-HDL cholesterol. This precipitation technique is the most frequently used method for HDL procedures.

R1 (diluted precipitant) 500 micro + 200 micro samples, mix, and allow to sit for 10 min at room temperature. Then centrifuge for 10 min at 4000 rpm. Finally, immediately separate off the clear supernatant and determine the cholesterol content using the CHOD-PAP method.

RESULTS

Age

There were no significant variations (P> 0.05) in age among control and patient communities in the study described, as indicated in Table 1. Figure 1 revealed that the average age of the patient group is slightly older than the average age of the control group. This change, however, is unrelated to hepatitis C virus infection.

Sex

According to Chi-squares, there is no statistically obvious variation (P> 0.05) between female and male ratios of the control and symptomatic groups. (Table 2 and Figure 2). Furthermore, the two groups of HCV patients and the control group are identical in size, suggesting that the gender difference is unrelated to hepatitis C virus patients.

Quantitative polymerase chain reaction (PCR)

The patient group is subjected to an HCV PCR test, with an average result of 956491.64 ±809304.4. (Table 3). Clarifying that the polymerase chain reaction (PCR) is a technique used specifically for HCV patients in which HCV particles can be counted and clearly explaining that HCV patients' PCR results

have extremely significant variance among patients. Furthermore, PCR was not necessary for the healthy group.

Creatinine

There was no obvious variation (P> 0.05) in creatinine levels between the control and patient groups (Table 4 and Figure 3). There was no noticeable difference in the kidney's major function test (creatinine).

Thyroid Stimulating Hormone (TSH)

Regarding thyroid stimulating hormone (TSH), all studies in our analysis discovered no difference in TSH levels between HCV-infected patients and healthy individuals. No statistically relevant relation existed between the control and patient groups (P> 0.05). (Table 5 and Figure 4).

liver function

Hepatic marker were represented by 7 clinical analyses. Bill, ALB, and ALK.PH. There is no obvious variation when comparing healthy and patient groups (P> 0.05). GGT, AST, ALT, and Bill, on the other hand. Significant variation existed between control and patient groups (P 0.05) (Table 6 and Figure 5).

Hematology

In the presented study, 6 clinical analyses representing hematology were assessed. WBC and HB were examined between healthy and infected groups and showed no obvious variation (P> 0.05). Platelets, INR, PT, and PC%, on the other hand, showed a clear variation (P 0.05) between healthy and infected groups (Table 7 and Figure 6).

Patients with HCV were included in the research. This study looks at the prevalence of anemia (lower RBC and hemoglobin levels) in HCV patients. Along with anemia and RBS, HCV-infected individuals have a lower platelet count than the reference value. WBC differential counts such as neutrophils, monocytes, lymphocytes, and eosinophils have been strongly linked with HCV positivity. It was discovered that HCV patients suffer from anemia due to lower hemoglobin content and RBC count.

α-Feto Protein

AFP shows an obvious variation (P> 0.05) between the healthy and patient groups (Table 8 and Figure 7).

Lipid Profile

Four clinical analyses of the lipid profile were evaluated. There was no significant difference in cholesterol, TG, or LDL across the tested communities (P> 0.05). In terms of HDL, there were clear variations between the control and infected patient communities (P> 0.05). (Table 9 and Figure 8). This discovery demonstrated that HCV infection is associated with alterations in lipid levels.

Blood glucose

FBS demonstrated a significant (P> 0.05) difference in the control and ill groups. In relation to controlling and patient, there is no significant change (P> 0.05) in Post.PBG (Table 10 and Figure 9).

Qualitative polymerase chain reaction (PCR)

After 3 and 6 months of therapy, there were significant changes (P 0.05) between negative and positive patients. After comparing 3 months and 6 months of therapy, there was no difference. (Table 11 and Figure 10).

DISCUSSION

HCV is a liver inflammation induced by the C virus. The virus can induce acute and chronic hepatitis, with symptoms varying from mild to life-threatening, including liver cirrhosis and malignancy. Hepatitis C virus is a blood-borne virus, and the majority of infections result from blood exposure via hazardous injecting practices, unsafe health care, and unscreened blood transfusions.

Verna *et al*. (2020) conducted a retrospective analysis of people with recovered HCV-related cirrhosis and found a substantial difference in disease severity between patients and controls. The clinical use of DAAs successfully treats hepatitis C virus infection and has resulted in a worldwide drop in its occurrence, which is consistent with the results of this study. Simple and easily available techniques based on clinical/biochemical indicators are necessary to assist physicians in assessing disease development in the liver. In this regard, albumin is a marker of liver synthesis; in our study, the ALT and AST scores are widely validated liver function measures. Our data suggest that the beneficial effect of viral elimination can assist HCCV patients in preventing liver disease development and improving liver function. In one trial, 642 cirrhotic individuals were treated with HCV elimination, and around 29% improved liver function as evaluated by the MELD score, which is helpful for quick prognosis.

Furthermore, after data extrapolation from four extensive studies utilizing DAAs, the importance of albumin levels as a predictor for disease improvement in the cirrhotic population was verified retrospectively (El-Sherif *et al*., 2018). These findings were approved in a second multicenter study of compensated and decompensated individuals administered by CHILD score. (Maan *et al*., 2016). Eliminating the detrimental component decreases liver inflammation and stiffness, especially in cirrhotic livers, and may result in decreased decompensating rates. (Krassenburg *et al*., 2021).

AFP is a well-studied biomarker. Serum AFP levels are significantly higher in individuals with compensated liver cirrhosis caused by HCV infection but without HCC. According to epidemiologic data, high AFP levels range from 10% to 43% in persons with HCV and compensated cirrhosis. Furthermore, in a cross-sectional study of 9800 patients, 2601 of whom had fatty liver disease (FLD), those with FLD had greater blood AFP levels than those without, per our findings**.** (Daniela *et al*.,2020).

Regarding hematological alterations, the current study indicated that when comparing baseline and follow-up data, the total blood picture parameters showed minimal modifications. Anemia, thrombocytopenia, and leucopenia were the most often reported side events, which may demand dosage modification in some individuals. (Hussein *et al*., 2022).

In this study, we noticed a gradual drop in lipid functions such as total cholesterol (TC), low-density lipoprotein (LDL), and triglyceride (TG), but high-density lipoprotein (HDL) increases, at an average level; these deregulations were much higher than in a demographically comparable HCV-free group. A substantial amount of evidence suggests an association between HCV and lipid deregulation. Encounter Corey *et al.* (2011) As compared to the healthy group, infected patients had significantly lower levels of LDL and TC, and HCV infection has been linked to variable fluctuations in lipid levels. Such dynamic lipid changes point to a direct link between the virus and lipid metabolism. A lack of awareness regarding the accurate timing or duration of infection and a lack of a suitable control group restricted previous studies. (Adeel *et al*., 2015) do not specify if increases in lipid levels are the result of HCV infection or if significant modifications existed prior to HCV infection. Our findings contribute considerably to current knowledge by revealing that certain alterations develop mostly following HCV infection, suggesting an indirect function for HCV infection in such changes.

Despite prior studies demonstrating a relationship between Hepatitis C with an increased hazard of CKD; (Park *et al.* 2018), In this prospective study, hepatitis C virus infection was related to an elevated risk of developing CKD and renal evolution in those with natural kidney function. The findings highlighted the importance of viral diagnosis in blocking CKD and maintaining renal function in the general population, which has prognostic and public health implications. As a result, serial serum creatinine measures were few, potentially underestimating the true prevalence of CKD. HCV can cause systemic inflammation as well as immune dysfunction, which can lead to glomerular damage. Additionally, In patients taking antiviral therapy, decreases in viral load were related to improved renal function, according to interventional investigations, demonstrating that HCV has an autonomous function in motivating renal impairment from the beginning of the illness. In clinical trials, HCV-infected diabetic patients who received DAA treatment and maintained a durable virologic response had better glycemic control and required fewer hypoglycemic drugs than those who did not. Given the metabolic, oxidative, and inflammatory disruption related to these illnesses, the effect of HCV infection on renal development in patients is likely to be concealed in part by the presence of substantial comorbidities. Further study is required to understand the pathophysiological processes of HCV-induced renal impairment in various early kidney illnesses and related comorbidities. Individuals infected with HCV were at a high risk of developing CKD. (Cheng *et al*., 2021).

CONCLUSION

The latest study indicates the influence of the Hepatitis C virus on blood parameters and body physiology in HCV patients. In addition, there was a significant genotype incidence among Sofosbuvir patients in our study.

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Table 1: Average ± SD of participant age for control and patient group

Group	Age
Control	39.93 ± 10.74
Patient	43.2 ± 10.54

Table 2: Sex ratio and Chi-square of control and patient group

Groups	Female	Male	X^2	P-Value
Control	6	9	0.6	0.439
Patient	6	9	0.6	0.439

Table 3: Average ± SD of Quantitative PCR result for the patient group before the present study

Group	PCR-HCV
Control	negative
Patient	956491.64 ± 809304.4

Table 4: Average ± SD of creatinine result for control and patient group

Group	Cr
Control	0.9 ± 0.13
Patient	1.18 ± 1.03

Table 5: Average ± SD of TSH hormone result for control and patient group

Group	TSH
Control	2.08 ± 0.8
Patient	2.11 ± 1.06

Table 6: Average ± SD of liver biomarker results for control and patient group.

Group	ALB	GGT	ALK.PH	AST	ALT	Bill. Total	Bill. Dir
Control	4.01 ± 0.33	27.06 ± 7.11	93 ± 19.81	17.86 ± 4.95	16.73 ± 3.92	0.71 ± 0.21	0.2 ± 0.06
Patient	4.1 ± 0.45	57.73 ± 57.9	88.26 ± 23.77	62.2 ± 45.88	59.2 ± 29.98	1.09 ± 0.7	0.44 ± 0.38

Table 7: Average ± SD of hematological parameters results for control and patient group.

Group	WBC	HB	Platelet	INR	PT	PC%
Control	5.88 ± 1.04	13.2 ± 1.04	316.2 ± 66.81	1 ±0.06	12 ± 1.62	100 ± 6.53
Patient	5.8 ± 1.42	13.04 ± 1.54	178 ± 55.75	1.08 ± 0.08	12.96 ± 1.01	91.12 ± 8.4

Table 8: Average ± SD of AFP (Tumor marker) for control and patient group

Group	AFP
Control	8.71 ± 4.71
Patient	5.45 ± 7.05

Table 9: Average ± SD of lipid profile results for control and patient group

Group	Cholesterol	TG	HDL	LDL
Control	145.53 ± 22.77	112.66 ± 21.16	47.46 ± 4.28	79.66 ± 7
Patient	150.06 ± 36.46	124.8 ± 54.79	38.46 ± 14.55	92.33 ± 21.9

Table 10: Average ± SD of blood sugar results for control and patient group

Group	FBS	Post.PBG
Control	79.26 ± 5.13	104.33 ± 5.72
Patient	94 ± 8.64	111.86 ± 24.33

Table 11: Average ± SD of PCR result after 3 and 6 months of treatment for the patient group.

Period	Positive	Negative	X^2	P-Value
3 Months	3	12	5.4	0.02
6 Months	3	12	5.4	0.02

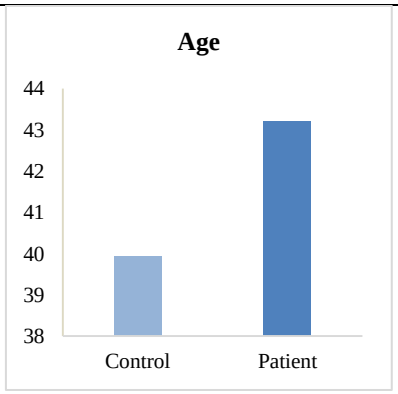

Figure 1: Column chart represents the age average for the control and patient group.

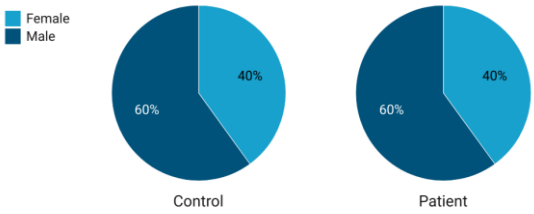

Figure 2: Pie chart representing the sex ratio of control and patient group.

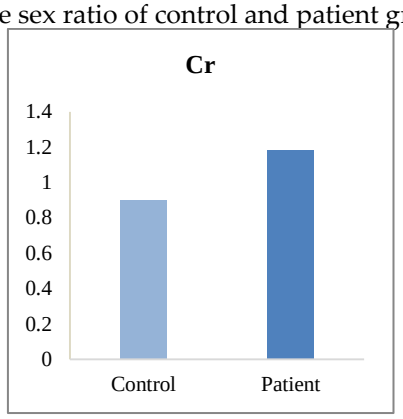

Figure 3: Column chart represents Cr average for control and patient group.

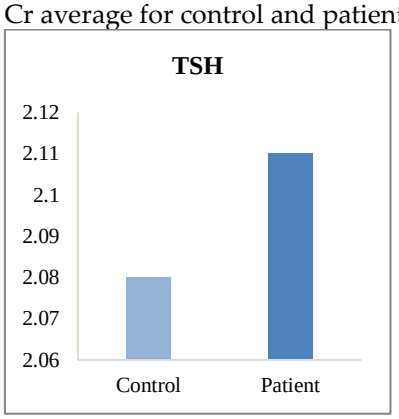

Figure 4: Column chart represents average TSH result for control and patient group.

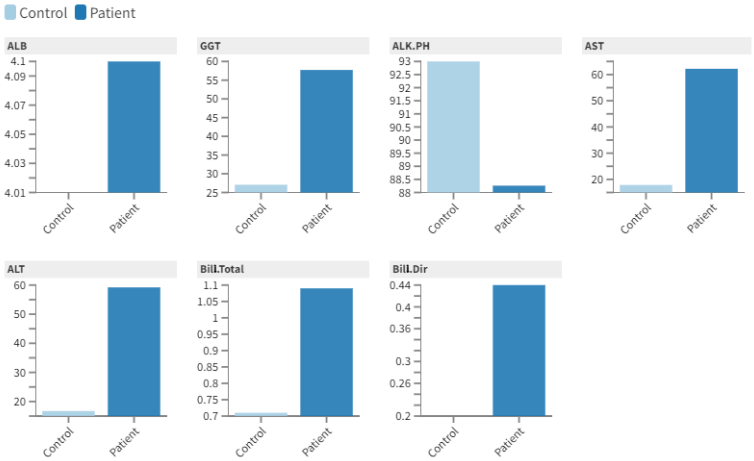

Figure 5: Column chart represents liver marker results for control and patient group.

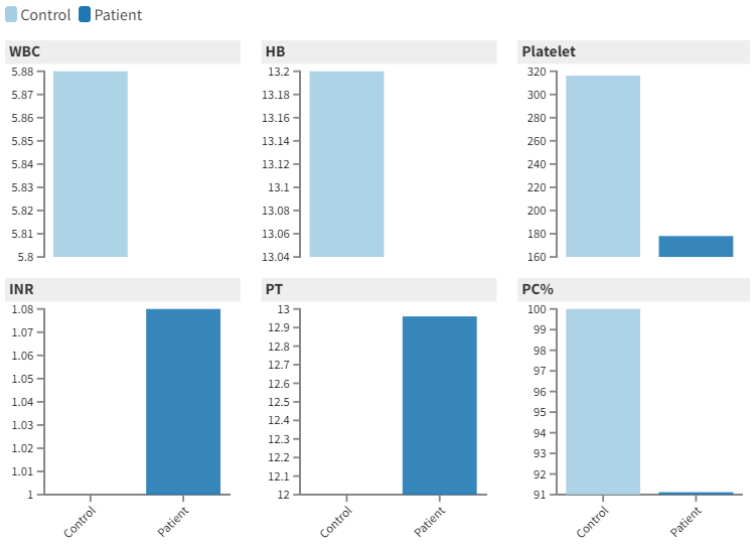

Figure 6: Column chart represents hematological biomarker results for control and patient group.

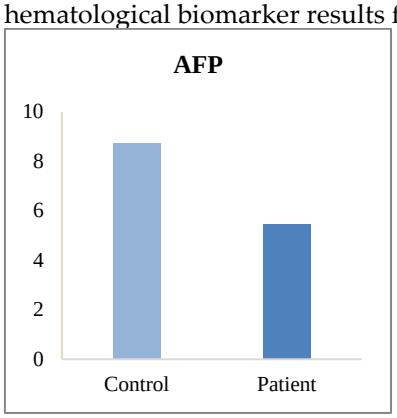

Figure 7: Column chart represents AFP results for control and patient group.

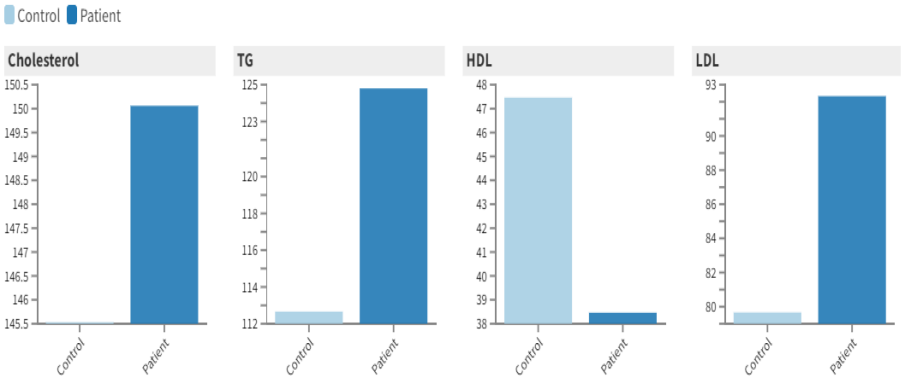

Figure 8: Column chart represents lipid profile results for control and patient group.

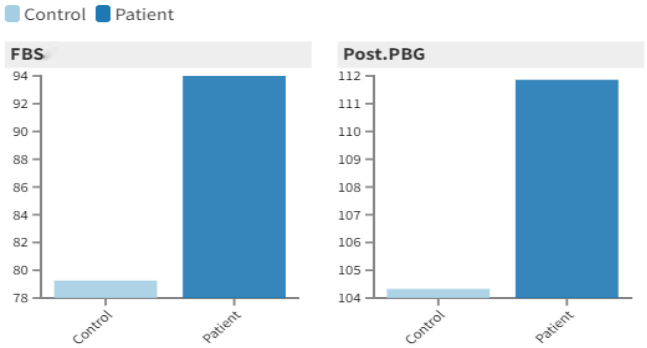

Figure 9: Column chart represents blood sugar results for control and patient group.

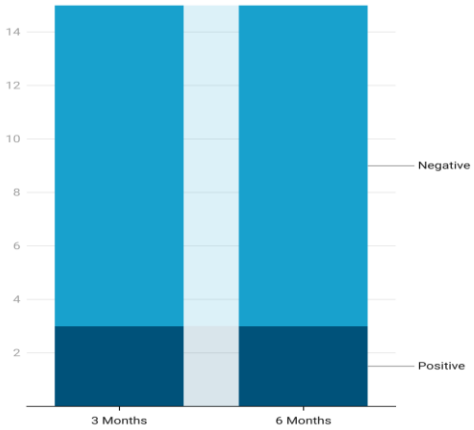

Figure 10: Stacked column chart represents qualitative PCR results for the patient group after 3 and 6 months.

الوظائف البيوكيميائية والفسيولوجية للمرضى المصريين المصابين بفيروس سي

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الملخص العربى

تعد الإصابة بفيروس سي مشكلة صحية عالمية ومعلوم أن فيروس سي سب ب شائع للإصابة بتليف الكبد الذي قد يؤدي للإصابة بسرطان الكبد ولتعريف هدف البحث تم قياس الوظائف الحيوية ودلالات أورام الكبد ، ومكونات الدم ، ووظائف الكلى، والدهون، وسكر الدم في فئتين من الناس : فئة مصابة بفيروس سي أخرى غير مصابة، وفي الدراسة الحالية تم معاينة الألبيومين والألكالين فوسفاتي ز والصفراء الكلية في الفئتين ولم يوجد تباين في النتائج ، وعلى النقيض ظهرت اختلافات بين الفئتين في بعض وظائف الكبد مثل جاما جي تي والصفراء المباشرة، وفيما يتعلق بالدهون والتي تشمل الكوليسترول والدهون الثلاثية والدهون ذات الكثافة المنخفضة(الضارة)والمرتفعة(النافعة)لم يظهر اختلاف بين الفئتين كما أن تحليل البيانات للدلالات البيوكيميائية والفيسيولوجية الخاصة بفيروس سي عامل أساسي لفهم طبيعة المرض وخطة علاج المصريين المصابين بفيروس سي.

الكلمات الاسترشادية :فيروس سي, الدلالات البيوكيميائية, الدلالات الفيسيولوجية, المصابون المصريون.