Effect of HCV treatment with DAAs on serum Intestinal Fatty Acid Binding Protein (I-FABP) as a marker of intestinal permeability in HCV/ HIV co-infected patients

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Research Article

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Abstract

Background: HCV and HIV co-infected patients develop cirrhosis more rapidly than HCV mono-infection. Intestinal injury and microbial translocation are postulated mechanisms for rapid progression of cirrhosis.

Aim: Study the effect of HCV treatment with DAAs on serum Intestinal Fatty Acid Binding Protein (I-FABP) as a marker of intestinal injury in HCV/ HIV co-infected patients and its relation to hepatic fibrosis. Comparing the level of I-FABP in HCV mono-infection and HCV/ HIV co-infection was a secondary aim.

Methods: I–FABP levels were measured in 50 non-cirrhotic HCV/HIV co-infected patients pre and post HCV treatment (SVR 12) and in 25 chronic HCV patients as a control group. Hepatic fibrosis was assessed by FIB4 score, APRI score and transient Elastography.

Results: HCV/ HIV co-infected patients had significantly higher levels of I-FABP compared to the HCV-mono-infected patients (P = 0.001). After HCV treatment in HCV/HIV co-infected patients, I-FABP level was significantly elevated (P <0.001) and was positively correlated to baseline FIB4 values and serum ALT level (r = 0.283, p value = 0.047) and (r= 0.340, P value = 0.016), respectively.

Conclusion: HCV/HIV co-infection is associated with significantly higher intestinal injury and subsequent hepatic fibrosis than HCV mono-infection. HIV infection is associated with intestinal epithelial injury and microbial translocation and may play a role in the persistence of systemic inflammation after HCV eradication.

Introduction

Elevated level of many blood markers indicative of pathological bacterial translocation or systemic inflammation have been detected in HIV-infected patients such as lipopolysaccharide and Intestinal fatty acid binding protein [1]. Cross-sectional transient Elastography data in HIV-infected patients have yielded high percent of advanced fibrosis in HIV-infected patients that may be due to direct hepatotoxic effect, dyslipidemia or insulin resistance associated with HIV infection, even without alcohol abuse or underlying viral hepatitis [2].

Data about the prevalence of HCV in persons living with HIV in Egypt is scarce. However, the prevalence of HIV among HCV-infected Egyptian patients was (0.66%) in one study [3].

HCV/HIV co-infected patients are more prone to accelerated progression of their hepatic fibrosis to cirrhosis [4]. One proposed mechanism is that co-infected persons have impaired intestinal barrier allowing translocation of bacteria into portal circulation with consequent hepatic and systemic inflammation [5].

Previous studies have measured markers of microbial translocation including I-FABP (marker of intestinal epithelial damage and is associated with microbial translocation) and found that these markers were

significantly elevated in HCV/HIV co-infected patients than in patients with HCV mono-infection [6, 7].

Understanding the association between HCV, HIV, microbial translocation and liver fibrosis may be beneficial in deciding interventions to reduce the harmful consequences of microbial translocation [7].

One study assessed the effect of HCV treatment with DAAs in HCV/HIV co-infected patients on microbial translocation and found improvement in severity of liver stiffness measurement and plasma inflammatory markers such as IL -18 and IL-8[8].

Objectives

We aimed to study the effect of HCV treatment with DAAs on serum Intestinal Fatty Acid Binding Protein (I-FABP) as a marker of intestinal injury in HCV/ HIV co-infected patients and its relation to hepatic fibrosis. Comparing the level of I-FABP in HCV mono-infection and HCV/ HIV co-infection was a secondary aim.

Patients And Methods

Patient's selection

This cross-sectional prospective study was conducted on patients with chronic HCV candidate for HCV treatment with DAAs directed to Kasr Al-Aini Viral Hepatitis Center (KAVHC) – Faculty of Medicine – Cairo University as one of the approved centers for HCV treatment during the period from March 2019 to April 2021.

The included patients were adults above the age of 18 years of both sexes, able to give informed consent and compliant to study procedures. Patients included in the study were non cirrhotic by FIB4 score to eliminate the effect of liver cirrhosis on I-FABP level. HCV infection was diagnosed by positive HCV antibody for more than 6 months and detectable HCV viremia. The recommended protocol for HCV treatment was Sofosbuvir and Daclatasvir for 12 weeks.

Exclusion criteria were patients refusing or unable to write the informed consent, pregnancy, breast feeding, history of chronic intestinal diseases as celiac disease or inflammatory bowel diseases and other inflammatory conditions, Patients having active intestinal infection or on antibiotic treatment for the previous 4 weeks, chronic liver diseases due to causes other than HCV infection, history of using NSAIDS for 2 weeks before visit and alcohol drinking > 4 drinks per week by a man or > 3 drinks per week by a woman).

Patients grouping

The enrolled patients were divided into two groups based on HIV infection as follow:

- Group I (25 patients): non-cirrhotic chronic HCV mono-infected patients.

- Group II (50 patients): non-cirrhotic chronic HCV/HIV co-infected patients.

Consent

A written informed consent was obtained from all patients. The study was performed in compliance with the ethics principles of the 1975 Declaration of Helsinki and its later amendments with Good Clinical Practice (GCP) guidelines.

Approval of the Institutional Review Board (IRB) of Kasr Alainy School of medicine, Cairo University was obtained for our study (D-43-2019) and for the umbrella project entitled “HCV prevalence among patients infected with HIV registered for HAART in Imbaba fever hospital in Cairo” (N-149-2018).

Methodology

Patients were subjected to thorough history-taking with special emphasis on presence of co-morbidities, special habits of medical importance and drug abuse. Blood samples were obtained for complete biochemical, serological and virological testing.

FIB4 and APRI scores were calculated. FIB-4 score was calculated using the following formula FIB-4 = Age [yrs] × AST [U/L] /platelet count [Plt ×〖10〗^9/L] × (ALT1/2[U/L]) [9]. APRI score was calculated using the following formula APRI = (AST/upper limit of normal x 100)/platelet count [10].

I-FABP testing was done by ELISA kit that uses Sandwich-ELISA method. The Micro ELISA strip plate provided in this kit has been pre-coated with an antibody specific to I-FABP. Standards or samples were added to the appropriate Micro ELISA strip plate wells and combined to the specific antibody. Then a Horseradish Peroxidase (HRP)- conjugated antibody specific for I-FABP was added to each Micro ELISA strip plate well and incubated. Free components were washed away. The TMB (Tetramethylbenzidine) substrate solution was added to each well. Only those wells that contain I-FABP and HRP conjugated I-FABP antibody will appear blue in color and then turn yellow after the addition of the stop solution.

The optical density (OD) is measured spectrophotometrically at a Wave length of 450 nm. The OD value was proportional to the concentration of I-FABP. Calculations of the concentration of I-FABP in the samples were performed by comparing the OD of the samples to the standard curve.

The reference range of the used kits was (< 2 ng/ml). Calibration of the I-FABP testing level was performed based on the I-FABP results of 25 healthy subjects as control group, the calibration range of the control group was (6.09 ± 1.90ng/ml).

Liver fibrosis and steatosis were assessment by **FibroScan®** (EchoSens, Paris, France) with the standard M probe and XL probe for obese patients. Measurements were performed through the intercostal spaces, where patients lying in the dorsal decubitus position with the right arm in maximal abduction. Measurements were performed after overnight fasting. Patients were further categorized into:

1. Non-significant fibrosis **(< F2)**

2. Significant fibrosis **(≥ F2)** [11].

Assessment of steatosis by CAP (Controlled Attenuation Parameter) and results were as categorized into the following:

- S0: no steatosis (0–237 dB/m).
- S1: mild steatosis(238–259 dB/m).
- S2: moderate steatosis (260–292 dB/m).
- S3: severe steatosis (≥ 293 dB/m) [12].

Laboratory workup, transient elastography and I-FABP testing were performed at baseline for the two patients groups and post HVC treatment (SVR 12) for group II.

Statistical Methods:

Data were coded and entered using the statistical package for the Social Sciences (SPSS) version 26 (IBM Corp., Armonk, NY, USA). Data was summarized using mean and standard deviation for quantitative variables and frequencies (number of cases) and relative frequencies (percentages) for categorical variables. Comparisons between groups were done using unpaired t test when comparing 2 groups and analysis of variance (ANOVA) with multiple comparisons post hoc test when comparing more than 2 groups [13].

For comparing categorical data, Chi square ($\chi 2$) test was performed. Exact test was used instead when the expected frequency is less than 5. For comparing categorical data measured before and after treatment McNemar Test was used [14]. Correlations between quantitative variables were done using Pearson correlation coefficient [15]. *P* values less than 0.05 were considered as statistically significant.

Results

Demographic characteristics of the studied two groups of patients (table 1)

The highest mean age was in HCV mono-infected group (47.76 ± 14.21 years) while in the HCV/HIV co-infected group (34.02 ± 11.12 years). Regarding gender distribution, male participants were higher in HCV/HIV co-infected groups (86%), while in HCV mono-infection group female participants percent was higher than male participants (56 versus 44%).

Regarding the risk factors for HCV and HIV infection acquisition in HCV/HIV co-infected group 82% of the studied patients were IV drug abusers, 40% had history of surgical intervention, 38% performed dental procedures and 10% of the patients received blood transfusion. Risky sexual behavior was denied by the studied patients in the HIV infected group.

Baseline characteristics of the studied two groups of patients (table 2)

Concerning laboratory parameters of the two groups, no statistically significant difference was noticed between the serum transaminases level and bilirubin levels in of both groups. While the mean HCV RNA PCR was **statistically significantly** higher in group II than group I with *P* value of 0.016.

Regarding I-FABP level, mean value was significantly higher in group II (11.76 ± 7.26) than group I (6.91 ± 1.25) with *P* value = 0.001.

Regarding the assessment of hepatic fibrosis, the mean stiffness value and significant fibrosis (≥ F2) were significantly higher in group II compared to group I with P value < 0.001. However, no statistically significant difference was detected between the two groups using the FIB4 and the APRI scores in assessment of hepatic fibrosis (p value 0.107 and 0.252, respectively). Concerning the steatosis assessment by CAP no statistically significant difference was detected between the two groups with p value of 0.711.

Regarding I-FABP level, the mean value was **significantly** higher in group II (11.76 ± 7.26) than group I (6.91 ± 1.25) with *P* value = 0.001. Regarding the I-FABP level in the control group the I-FABP level in the HCV group was comparable to the control group while significantly higher in group II than the control group.

Group II characteristics pre and post HCV treatment (table 2)

Mean I-FABP level was **significantly** higher after HCV treatment (20.11 ± 7.30) than before treatment (11.76 ± 7.26) with *P* value **< 0.001**.

Comparison of laboratory parameters before and after treatment revealed that mean platelets count after HCV treatment (225.28 **±** 60.96) was significantly higher than the mean count before treatment (219.53 **±** 58.09) with *P* value of 0.016. Mean AST and ALT levels after treatment were (27.30 **±** 8.99) and (27.76 **±** 10.99) respectively and both values were significantly lower than the mean values before treatment which were (39.46 **±** 22.72) and (62.14 **±** 15.96), respectively with *P* value of < 0.001 for both.

Concerning assessment of liver fibrosis, mean FIB4 and APRI scores after HCV treatment were 0.82 **±** 0.45 and 0.25 **±** 0.11, respectively which were **significantly** lower than the mean values before treatment 0.96 **±** 0.62 and 0.40 **±** 0.30 respectively with *P* value = 0.019 for FIB4 and < 0.001 for APRI score, respectively.

Correlation between serum I-FABP and the studied parameters in group II pre and post HCV treatment (table 3)

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Only significant correlation between the baseline FIB4 and ALT level and the post treatment I-FABP level (r = 0.283, p value = 0.047) and (r = 0.340, *P* value = 0.016) respectively as shown in figure (1) and (2).

Concerning the correlation between post-treatment parameters in the HCV/HIV co-infection group and the I-FABP value before and after treatment revealed only **significant** positive correlation between post-treatment bilirbin level and the pre-treatment I-FABP level (r = 0.286, p value = 0.044).

Discussion

The current study revealed higher serum I-FABP level detected in the HCV/HIV co-infected patients than HCV mono-infected patients. Previous studies noticed the same results [6&16]. This could be explained by the additive effect of both viruses on intestinal mucosal damage. This also was explained by mucosal CD + 4 T cell depletion due to HIV infection or may be due to more liver disease progression in co-infected patients [6, 16&17].

In our HCV mono-infected group, the serum I-FABP level was higher than the reference range of the kits but not significantly higher than healthy control participants. Our results agree with previous similar study by Wurcel where mean I-FABP level was 294 pg/ml and 386 pg/ml in HCV patients and control group, respectively and the difference was not statistically significant [16]. In contrary, Reid et al. found significant elevation of the serum I-FABP level in HCV mono-infected patients compared with control group. This may be explained by the difference in the study population, as the latter study included, cirrhotic patients and we excluded cirrhotic patients in our study as elevated I-FABP level may be related to enterocytes damage and microbial translocation that is known to occur in liver cirrhosis [18].

In our exploratory analysis, baseline FIB4 score was positively correlated with the post-treatment serum I-FABP levels in HCV/HIV co-infected patients. This positive correlation suggests a relationship between increased I-FABP and increased liver disease severity. Similar results were confirmed by previous studies Wurcel and French et al., [16&17].

Another potential explanation for the observed statistical correlation between I-FABP in HCV/HIV co-infection and FIB4 score is the effect of anti-retroviral therapy (ART) on the liver in this group. Recent ART-related clinical syndromes such as NAFLD and non-cirrhotic portal hypertension have increased in patients on ART and observational studies propose long-term ART-related hepatic injury [19].

Significant improvement of the elevation of serum transaminases and improvement of platelet count in HCV/HIV co-infected patients after HCV eradication that was noted in our study is in concordance with the study by Brochado-Kith et al., who concluded that at the end of follow-up after all-oral DAA therapy, HCV/HIV-coinfected patients exhibited a significant decrease (q-value < 0.05) in AST and ALT and substantial increase in plateletscount was observed [20].

Regarding improvement of hepatic fibrosis in HCV/HIV co-infected patients after HCV treatment with DAAs, the significant improvement in the FIB4 and APRI scores could be explained by the amelioration of

HCV-induced necro-inflammation and fibrosis [21&22&23]. However, this improvement was not reflected on the results of transient Elastography and steatosis measured by CAP. This may be due to confounding effects of ART that may induce hepatic steatosis that consequently affects stiffness and steatosis measurements.

I-FABP level was significantly higher after HCV treatment in HCV/HIV co-infected patients. This may imply that HCV eradication on the liver has potential advantages on the liver (improvement in platelets count, albumin, serum transaminases levels and hepatic fibrosis), but the enterocyte permeability may persist or even worsen after HCV eradication. Studies have found that despite achieving SVR in HCV infected patients monocyte activation (part of systemic inflammation caused by bacterial translocation) may persist [24&25].

Our results are contrary to Medrano et al who found no significant change in I-FABP after HCV treatment (p value = 0.29). This difference may be due to choosing patients with advanced cirrhosis (LSM ≥ 25 kPa, or HVPG ≥ 10 mmHg, or CTP ≥ 7, or prior history of liver decompensation) which may affect baseline I-FABP level in contrast to our patients who were non cirrhotic [8].

Conclusion

In our study HCV/HIV co-infected patients had significantly higher I-FABP level in contrast to HCV mono-infected patients who had I-FABP level that was comparable to the control group. This may imply that HCV is not the primary driver of impaired permeability in co-infected patients and this is further confirmed by lack of improvement of I-FABP after HCV eradication in co-infected patients. Furthermore, advanced baseline liver Fibrosis (FIB4 score) was correlated to with higher degree of intestinal injury after HCV treatment with DAAs.

These data provide a starting point for future longitudinal studies looking at how markers of intestinal health and permeability can help in monitoring the natural history of some diseases and the efficacy of therapy.

Declarations

Approval of the Institutional Review Board (IRB) of Kasr Alainy School of medicine, Cairo University was obtained for study conduction and publication (D-43-2019) under the umbrella project entitled “HCV prevalence among patients infected with HIV registered for HAART in Imbaba fever hospital in Cairo” approved by cairo university (N-149-2018).

Consent for publishing not applicable

Data and materials are available. please contact Lamiaaalsehemy@kasralainy.edu.eg

None of the authors has conflict of interests.

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Authors contributions to work: Hanan Abdel-Haleem and Gamal esmat principel investigators,Marwa khairy and Mahmoud abdo revision of manuscript,Ahmed cordie data collection,Aisha El sharkawy data analysis and revision of tables, Marwa El sharkawyclinical pathological measurement of I-FABP,shereen Abdel Alem performed fibroscan,Lamiaa Al Sehemy wrote the manuscript and interviewed the patients

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Tables

Table (1): Demographic characteristics of the studied population

		Group I HCV only (n=25)		Group II HCV/HIV (n=50)		P value
Age (mean ± SD)		47.76 ±14.21		34.02 ±11.12		**<0.001**
		Count	**%**	**Count**	**%**	
Gender	**Male**	11	44.0%	43	86.0	**<0.001**
	Female	14	56.0%	7	14.0	
Smoking		7	28.0%	41	82.0%	**<0.001**
IV drug abuse		0	0.0%	41	82.0%	**<0.001**

Table (2): Baseline characteristics of the studied two groups of patients and post treatment follow up

		Group I HCV (n=25)	Group II HIV/HCV (n=50)		P1 value	P2 value
			pre HCV treatment	Post HCV treatment		
AST(<35 IU/ml)		39.04±25.04	39.46±22.72	27.30±8.99	0.942	**< 0.001**
ALT (<35 IU/ml)		57.92±71.10	62.14±50.96	27.76±10.99	0.769	**< 0.001**
HCV RNA PCR		0.772±0.1004	1.595±1.89	Undetectable	**0.016**	-
I-FABP reference range (<2 ng/ml)		6.91±1.25	11.76±7.26	20.11±7.30	**0.001**	**< 0.001**
I-FABP control range (6.09 ± 1.90ng/ml)		6.91±1.25 (p = 1.00)	11.76±7.26 **(p <0.001)**	20.11±7.30	**	**< 0.001**
Transient Elastography (kPa)		4.42±0.44	5.51±1.26	5.48±2.04	**<0.001**	0.928
Fibrosis Stages	**<F2 n (%)**	25 (100.0%)	41 (82.0%)	43 (86.0%)	**0.025**	0.727
	≥ F2 n (%)	0	9 (18.0%)	7 (14.0%)		
CAP (dB/m)		196.84±39.06	212.92±48.75	210.10±48.44	0.156	0.634
Steatosis grades	**<S2 n (%)**	23 (92.0%)	44 (88.0%)	44 (88.0%)	0.711	1
	≥ S2 n (%)	2 (8.0%)	6 (12.0%)	6 (12.0%)		
FIB4		1.21±0.57	0.96±0.62	0.82±0.45	0.107	**0.019**
APRI score		0.50±0.45	0.40±0.30	0.25±0.11	0.252	**< 0.001**

*P1 value difference between group I and group II pre HCV treatment

P2 value difference between pre and post HCV treatment in group II

** *P* value for group I with (calibration) control is 1.000 and for group II with calibration (control) **<0.001**

Abbreviations: APRI= AST to Platelet Ratio Index, CAP = Controlled Attenuation Parameter, dB/m= decibel per minute, FIB4= Fibrosis-4, kPa= kilo pascal, n=number, ng=nanogram

Correlation between serum I-FABP and the studied parameters in group II pre and post HCV treatment (table 3)

Baseline parameters	Pre-treatment I-FABP level		Post-treatment I-FABP level	
	Pearson Correlation (r)	P value	Pearson Correlation (r)	P value
AST (<35 IU/ml)	0.072	0.618	0.216	0.132
ALT (<35 IU/ml)	0.064	0.660	0.340	**0.016**
FIB4	0.113	0.436	0.283	**0.047**
APRI score	0.063	0.662	0.245	0.087
Fibroscan (kpa)	-0.090-	0.535	0.035	0.808
CAP (dB/m)	-0.067-	0.646	-0.063	0.666
HCV RNA PCR	-0.040-	0.784	0.085	0.556
CD4 count (500-1200 cells/mm^3)	-0.096-	0.508	0.175	0.223
HIV RNA PCR	-0.170-	0.243	-0.254	0.078

Post treatment parameters	Pre-treatment l-FABP level		Post-treatment l-FABP level	
	Pearson Correlation	P value	Pearson Correlation	P value
Bilirubin total (0.3-1.2mg/dl)	0.286	**0.044**	0.018	0.900
AST (<35 IU/ml)	0.006	0.967	0.047	0.748
ALT (<35 IU/ml)	0.100	0.489	0.195	0.176
FIB4	0.068	0.638	0.233	0.104
APRI	0.140	0.331	0.219	0.126
Fibroscan (kpa)	-0.066-	0.649	-0.070-	0.629
CAP (dB/m)	-0.030-	0.834	-0.077-	0.594

Figures

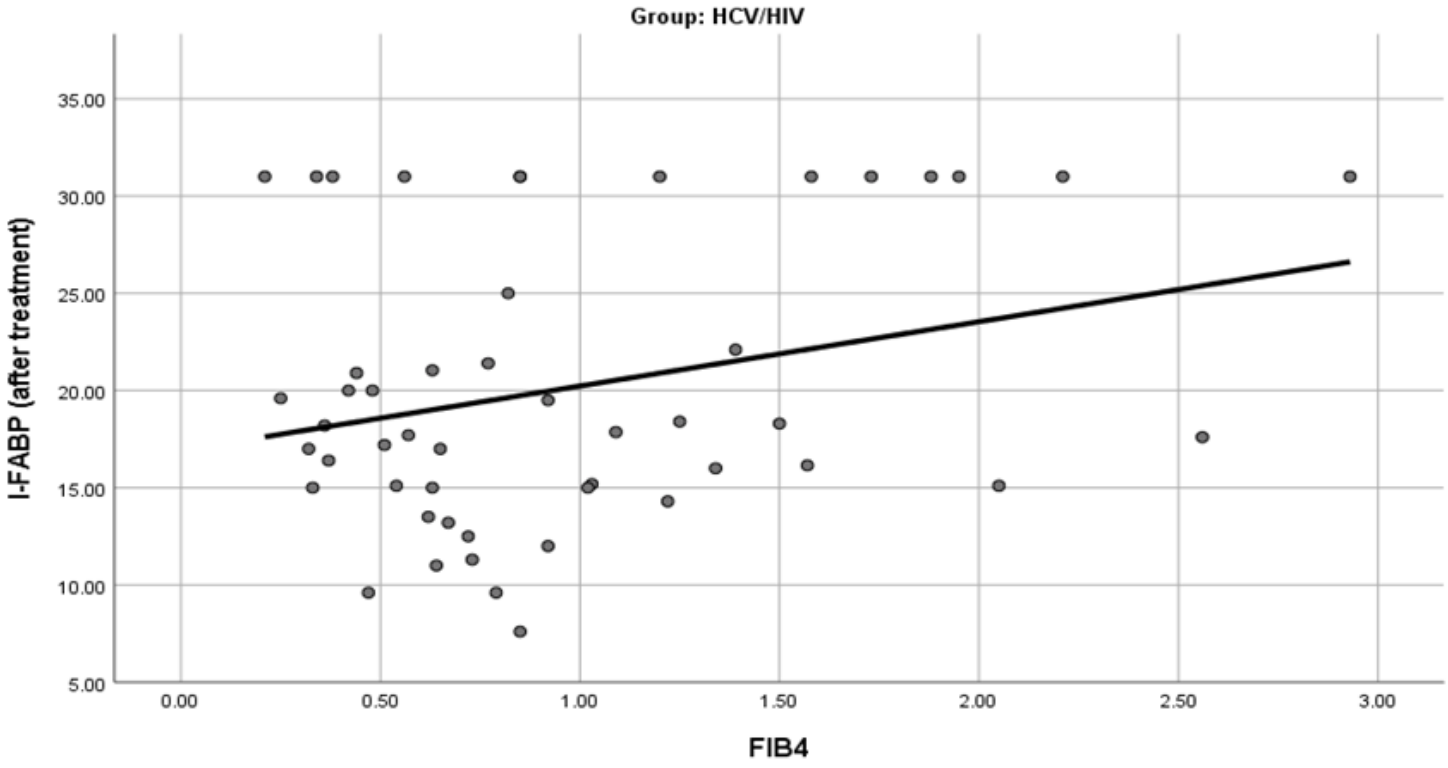

Figure 1

Significant positive correlation between baseline FIB4 score with post treatment I-FABP level in HCV/HIV co-infected group (r = 0.283, p value = 0.047)

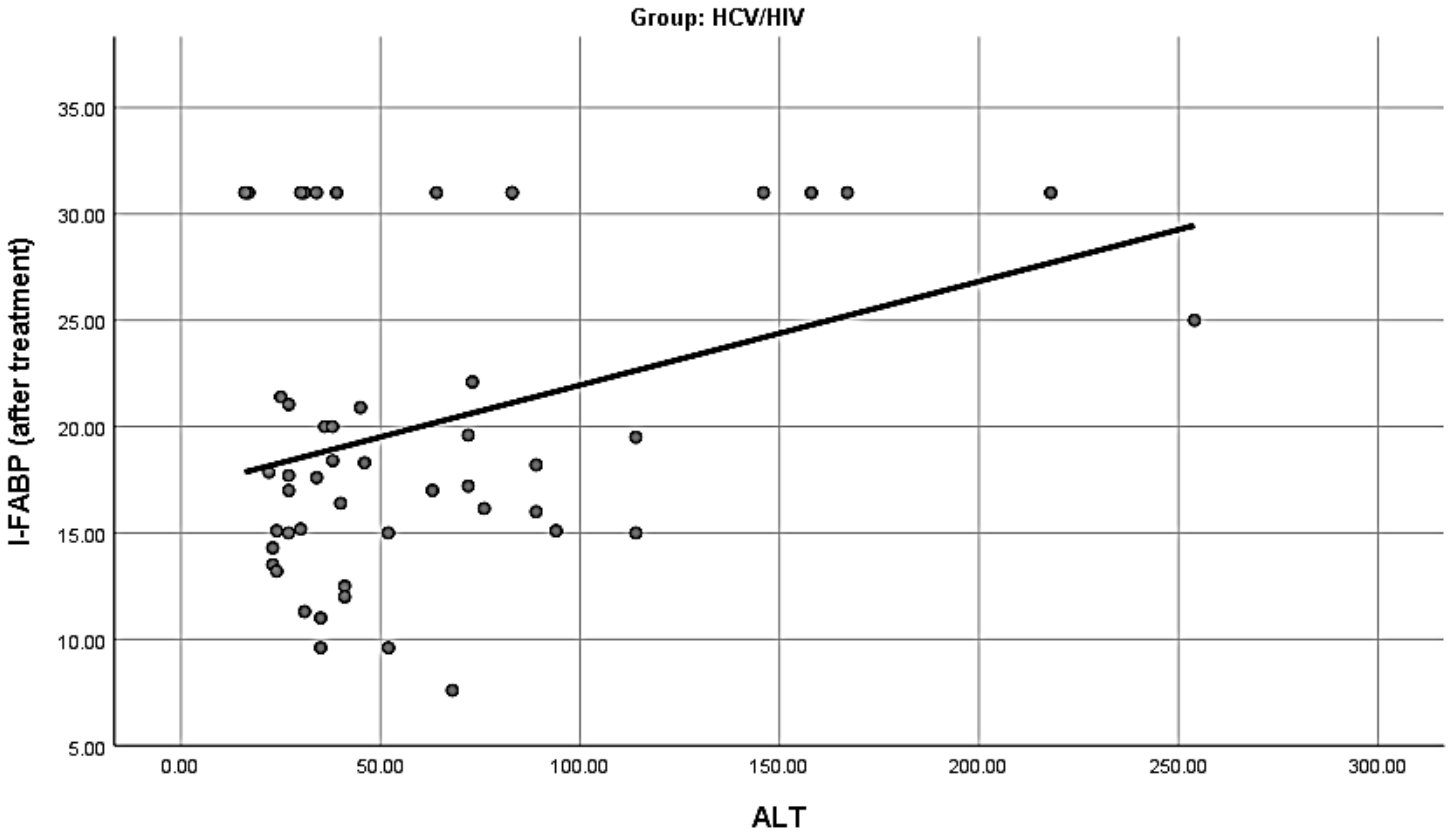

Figure 2

Significant Positive correlation between the baseline ALT and post treatment I-FABP level in HCV/HIV co-infected group (r= 0.340, p value = 0.016)