Assessment of the nutritional status and serum leptin level of hepatitis C virus-infected Egyptian patients with and without schistosomal hepatic periportal fibrosis

Said A. Ooda[a], Mostafa A. Mohamed[b], Maher A. Nabi[c], Wael M. Lotfy[b], Basem A. Alhabet[b]

Departments of [a]Internal Medicine, [b]Parasitology, [c]Biochemistry, Medical Research Institute, Alexandria, Egypt

Correspondence to Said A. Ooda, MD, Department of Internal Medicine, Medical Research Institute, 165 Horreya Avenue, Hadara, Alexandria, Egypt
Tel: +20 342 82331/+20 428 2373;
Fax: +20 342 83719;
e-mail: drsaidooda@gmail.com

Received 12 December 2015
Accepted 14 April 2016

Egyptian Journal of Obesity, Diabetes and Endocrinology
2016, April:36–45

Background
Schistosomiasis has been a major public health problem in Egypt. Moreover, Egypt has been widely regarded as having the highest recorded prevalence of hepatitis C virus (HCV) in the world. Malnutrition is prevalent in all forms of liver disease.

Objective
The aim of the work was to assess the nutritional status of HCV-infected patients with and without schistosomal hepatic periportal fibrosis.

Patients and methods
This study was carried out on 93 men. A total sample size of 93 patients was taken. The patients were divided into three groups of 31 each: group I included 31 patients having HCV; group II included 31 patients having mixed schistosomal hepatic periportal fibrosis and HCV; and group III included 31 healthy controls. Serum leptin was measured. Abdominal ultrasonography was performed to all participants to detect the degree of fibrosis according to the WHO scoring system. Nutritional assessment was carried out using anthropometric measurements. Body fat content was measured using bioelectrical impedance analysis.

Results
This study showed that the fat content was higher in group I than in group II and was higher in controls than in patients. Serum leptin level was significantly higher in group II than in group I.

Conclusion
Affection with HCV and/or schistosomal hepatic periportal fibrosis affects the nutritional status of the individuals affected, with more pronounced nutritional derangement in patients with both diseases.

Keywords:
bioelectrical impedance analysis, hepatitis C, leptin, periportal fibrosis

Egyptian Journal of Obesity, Diabetes and Endocrinology April:36–45

2356-8062

Introduction

Schistosomiasis has been a major public health problem in Egypt [1]. It affects child development and adult productivity. Schistosomiasis continues to be a significant cause of morbidity and mortality. The WHO considers schistosomiasis as second only to malaria in socioeconomic importance worldwide and the third more frequent parasitic disease in public health importance [2].

Beginning in the 1950s and continuing until the 1980s, the Egyptian Ministry of Health conducted large campaigns using the standard treatment at that time, tartar emetic, as community-wide therapy, not properly sterilized, and thus transferred traces of blood and blood-borne pathogens from human to human. As a result, this massive effort to control one health problem resulted in the creation of another, as hepatitis C virus (HCV) was spread through the intravenous injections. Indeed, this is estimated to be the largest known iatrogenic transmission of blood-borne infections in the history of the world [3].

With high prevalence rates for both HCV and schistosomiasis, it is inevitable that Egypt has a large number of humans with both diseases. Having both is more damaging to the liver and is associated with higher mortality rates than that having just one [4].

Nutrition status is recognized as a predictor of morbidity and mortality in patients with advanced liver disease [5]. The liver is an important regulator of metabolism, storage, synthesis, and absorption of nutrients. Accordingly, the severity of malnutrition

increases with decreases in liver function. Malnutrition is prevalent in all forms of liver disease: from 20% in compensated liver disease to more than 80% in those patients with decompensated liver disease [6]. Many patients have subtle changes such as fat-soluble vitamin deficiency, altered cell-mediated immune function, anemia from iron, folate or pyridoxine deficiency, and minimal loss of muscle mass. Patients with end-stage liver disease have muscle wasting, decreased fat stores, and cachexia [7].

Leptin, the adipocyte-derived protein product, is involved in appetite regulation and obesity through central effects at the hypothalamus. Leptin is related to amount of body fat (BF) [8].

Bioelectrical impedance analysis (BIA) is a commonly used method for estimating body composition, and, in particular, BF. Since the advent of the first commercially available devices in the mid-1980s, the method has become popular owing to its ease of use, portability of the equipment, and its relatively low cost compared with some of the other methods of body composition analysis. It is familiar in the consumer market as a simple instrument for estimating BF. BIA actually determines the electrical impedance, or opposition to the flow of an electric current through body tissues, which can then be used to calculate an estimate of total body water [9,10].

Aim of the study

The aim of this study was to evaluate the nutritional status of HCV-infected patients in association with schistosomal hepatic periportal fibrosis.

Patients and methods

This study was carried out on 93 men. The men were chosen because they have the same fat percentage. They were recruited from patients attending the Hepatology Department Clinic of Medical Research Institute, Alexandria University. All participants were asked to freely volunteer to the study and informed written consent was obtained before inclusion in the study, according to the ethical guidelines of Medical Research Institute, Alexandria University. A total sample size of 93 patients was taken. The study included three groups: group I, which included 31 patients with HCV; group II which included, 31 patients with mixed schistosomal hepatic periportal fibrosis and HCV; and group III, which included 31 healthy controls. Their ages ranged from 20 to 65 years.

All participants were subjected to the following and then assigned to the corresponding group.

(1) History and clinical examination
   (a) An information sheet was prepared for each patient.
   (b) Full history taking and thorough clinical examination were carried out.
(2) Abdominal ultrasonography to detect the degree of periportal fibrosis of schistosomiasis according to the WHO scoring system [11].
(3) Parasitological examination
   (a) Stool examination using sedimentation and Kato-Katz techniques [12].
   (b) Serology for schistosomiasis using the enzyme-linked immunosorbent assay technique [13].
(4) Nutritional assessment
   (a) Anthropometric measurements
      (i) Height.
      (ii) Weight.
      (iii) BMI [14].

BMI (kg/m^2)	Nutritional status
<16	Severe malnutrition
16–16.99	Moderate malnutrition
17–18.49	Mild malnutrition
18.5–24.9	Normal
25–29.9	Overweight
30–34.9	Obese class 1
35–39.9	Obese class 2
≥40	Obese class 3

      (iv) Waist and hip circumference.
      (v) Waist–hip ratio.
   (b) Body composition measurement

BF was measured using BIA. The BF loss monitor OMRON HBF-306 C (Omron Healthcare, Lake Forest, Illinois, USA) device was used. This measurement is performed after entering the age, height, and sex into the device and then the patient holds the device with his hands to calculate the fat percentage. The Omron BF loss monitor may underestimate BF by about 3% in comparison with measurement taken using calipers, as reported by the manufacturer [15].

In men, BF percentage below 13% is considered below average [16].

(5) Biochemical examination.

Blood sample was drawn and the following investigations were performed.

   (a) HCV infection was diagnosed by the presence of HCV antibodies using the enzyme-linked immunosorbent assay technique and confirming that using PCR for HCV.

Table 1 Age distribution among the studied groups

Age	Group I (*N* = 31)	Group II (*N* = 31)	Group III (*N* = 31)	*F*	*P*-value
Mean ± SD	43.65 ± 10.63	46.19 ± 8.88	41.23 ± 8.61	2.158	0.122
Minimum–maximum	20.0–65.0	25.0–59.0	28.0–65.0		

F, *F*-test (analysis of variance); group I, HCV; group II, HCV + schistosomiasis; group III, control; HCV, hepatitis C virus.

(b) Complete blood count.
(c) Liver enzymes [alanine transaminase (ALT), aspartate transaminase (AST)].
(d) Serum albumin.
(e) Lipid profile (LDL, HDL, and total cholesterol, triglycerides).
(f) Serum leptin [8].

The results were tabulated and analyzed according to the appropriate biostatistical methods.

Table 2 Clinical presentation of the studied groups

Symptoms	Group I (*N* = 31) [*n* (%)]	Group II (*N* = 31) [*n* (%)]	χ^2	*P*-value
Abdominal pain	15 (48.4)	17 (54.8)	0.258	0.611
Constipation	3 (9.7)	6 (19.4)	1.170	^{FE}P = 0.473
Hyperacidity	15 (48.4)	9 (29.0)	2.447	0.118
Regurgitation	7 (22.6)	6 (19.4)	0.097	0.755
Piles	2 (6.5)	0 (0.0)	2.067	^{FE}P = 0.492
Bleeding per gum	0 (0.0)	1 (3.2)	1.016	^{FE}P = 1.000

FE, Fisher's exact test; group I, HCV; group II, HCV + schistosomiasis; HCV, hepatitis C virus.

Results

Statistical analysis of the data

Data were fed into the computer and analyzed using SPSS software (version 20.0; SPSS Inc., Chicago, Illinois, USA) package for the social science, version 20.0. Qualitative data were described using number and percentage. Quantitative data were described using range (minimum and maximum), mean, SD, and median. Comparison between different groups as regards categorical variables was made using the χ^2-test. When more than 20% of the cells have expected count less than 5, correction for χ^2 was conducted using Fisher's exact test or Monte Carlo correction. The distributions of quantitative variables were tested for normality using the Kolmogorov–Smirnov test, Shapiro-Wilk test, and D'Agstino test. In addition, histogram and QQ plot were used for vision test. If it revealed normal data distribution, parametric tests were applied. If the data were abnormally distributed, nonparametric tests were used. For normally distributed data, comparisons between the studied groups were made using *F*-test (analysis of variance) and post-hoc test (Scheffe). For abnormally distributed data, the Kruskal–Wallis test was used to compare the studied groups and pair-wise comparison was assessed using the Mann–Whitney test. Significance test results are quoted as two-tailed probabilities. Significance of the obtained results was judged at the 5% level.

Age distribution

There was no statistical difference between the three studied groups with regard to age (Table 1).

Clinical presentation

There was no statistical difference between the studied groups with regard to the clinical presentation (Table 2).

Anthropometric measurements

Table 3 shows the anthropometric data of the different studied groups.

There was no statistical difference between the three studied groups with regard to height.

There was a significant statistical difference between the three studied groups with regard to weight. The highest mean weight was seen in controls and the lowest mean weight was seen in group II. Pair-wise comparison test denoted that there was a difference in the mean weight, being lower in group II compared with group III.

There was a significantly high statistical difference between the three studied groups with regard to waist circumference. The highest mean waist circumference was seen in controls and the lowest mean waist circumference was seen in group II. Pair-wise comparison test denoted that there was a difference in the mean waist circumference; it was lower in group I compared with group III and lower in group II compared with group III.

There was a significant statistical difference between the three studied groups with regard to hip circumference. The highest mean hip circumference was seen in controls and the lowest mean hip circumference was seen in group I. Pair-wise comparison test denoted that there was a difference in the mean hip circumference, being lower in group I compared with group III.

There was a significantly high statistical difference between the three studied groups with regard to waist–hip ratio. The highest waist–hip ratio was seen in controls and the lowest waist–hip ratio was seen in group II. Pair-wise comparison test denoted that

there was a difference in the waist–hip ratio; it was greater in group I compared with group II, lower in group I compared with group III, and lower in group II compared with group III (Fig. 1).

There was a significant statistical difference between the three studied groups with regard to BMI. The highest BMI was seen in controls and the lowest BMI was seen in group II. Pair-wise comparison test denoted that there was a difference in the BMI, being lower in group II compared with group III (Fig. 2).

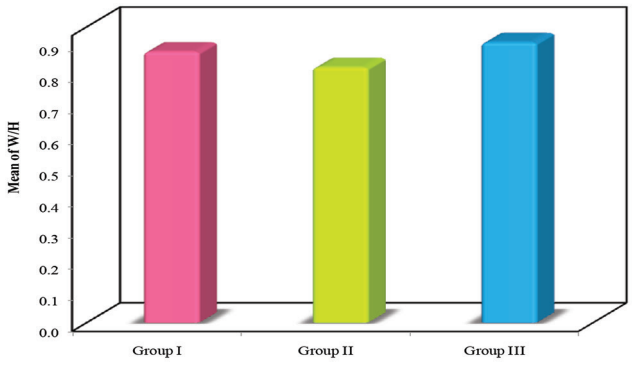
Ultrasound findings

Table 4 shows the ultrasound findings between the studied groups.

There was a significantly high statistical difference between the three studied groups. Pair-wise comparison test denoted that there was a difference in the liver structure between groups II and III as well as between groups II and III.

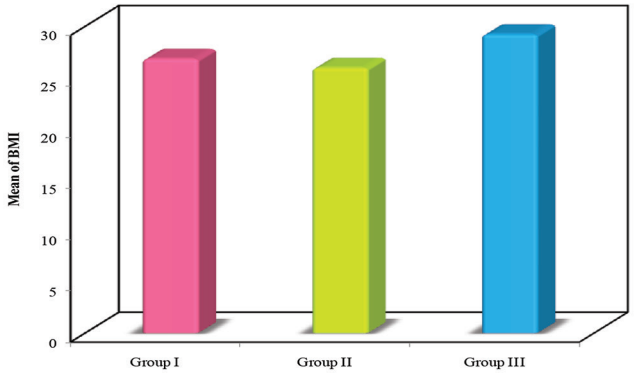
There was no statistical difference between the three studied groups with regard to right lobe liver diameter.

Figure 1



Comparison between the studied groups with regard to W/H. W/H, waist–hip ratio.

Figure 2



Comparison between the studied groups with regard to BMI.

Table 3 Anthropometric data of the different studied groups

Anthropometric data	Group I (*N* = 31)	Group II (*N* = 31)	Group III (*N* = 31)	*F*	*P*-value
Height (cm)					
Mean ± SD	172.023 ± 5.89	170.71 ± 7.57	172.26 ± 5.37	0.603	0.550
Minimum–maximum	160.0–188.0	155.0–189.0	160.0–182.0		
Weight (kg)					
Mean ± SD	79.23 ± 16.26	75.35 ± 11.97	87.35 ± 12.77	6.114*	0.003*
Minimum–maximum	57.0–120.0	55.0–102.0	64.0–111.00		
Pair-wise comparison		II–III**			
Waist (cm)					
Mean ± SD	91.65 ± 8.50	88.29 ± 5.14	100.87 ± 8.92	22.153*	<0.001*
Minimum–maximum	77.0–111.0	80.0–99.0	85.0–118.0		
Pair-wise comparison	I–III**, II–III**				
Hip (cm)					
Mean ± SD	105.87 ± 8.83	107.23 ± 5.59	111.71 ± 7.92	5.050*	0.008*
Minimum–maximum	90.0–125.0	98.0–122.0	97.0–126.0		
Pair-wise comparison	I–III*				
W/H					
Mean ± SD	0.87 ± 0.04	0.82 ± 0.02	0.90 ± 0.04	43.247*	<0.001*
Minimum–maximum	0.78–0.96	0.77–0.89	0.79–0.97		
Pair-wise comparison	I–II***, I–III***, II–III***				
BMI (kg/m^2)					
Mean ± SD	26.81 ± 5.10	25.94 ± 3.37	29.21 ± 3.79	5.1753*	0.008*
Minimum–maximum	20.20–38.70	19.0–32.70	23.20–37.60		
Pair-wise comparison	II–III*				

Pair-wise comparison was made using the post-hoc test (Scheffe). *F*, *F*-test (analysis of variance); group I, HCV; group II, HCV + schistosomiasis; group III, control; HCV, hepatitis C virus; W/H, waist–hip ratio. *Statistically significant at $P \leq 0.05$. **Statistically significant at $P \leq 0.01$. ***Statistically, significant at $P \leq 0.001$.

There was a significantly high statistical difference between the three studied groups with regard to portal vein diameter. Pair-wise comparison test denoted that there was a difference in the portal vein diameter; it was lower in group I compared with group II, greater in group I compared with group III, and greater in group II compared with group III.

In group II, 74.2% of patients had grade I liver fibrosis and 25.8% had grade II liver fibrosis. Pair-wise comparison test denoted that there was a difference in the degree of liver fibrosis between groups I and III as well as between groups II and III.

There was no statistical difference between the three studied groups.

There was a significantly high statistical difference between the three studied groups with regard to spleen diameter. Pair-wise comparison test denoted that there was a difference in the spleen diameter; it was greater in group I compared with group II and greater in group II compared with group III.

Complete blood picture

Table 5 shows the complete blood picture of the studied groups.

There was a significantly high statistical difference between the three studied groups with regard to red blood cell count. Pair-wise comparison test denoted that there was a difference in the red blood cell count; it was greater in group I compared with group II, lower in group I compared with group III, and lower in group II compared with group III.

There was a significantly high statistical difference between the three studied groups with regard to hemoglobin concentration. Pair-wise comparison test denoted that there was a difference in the hemoglobin concentration; it was greater in group I compared with group II, lower in group I compared with group III, and lower in group II compared with group III.

There was no statistical difference between the three studied groups with regard to white blood cell count. Pair-wise comparison test denoted that there was a difference in the white blood cell count; it was greater in group I compared with group II and lower in group II compared with group III.

There was a significantly high statistical difference between the three studied groups with regard to platelet count. Pair-wise comparison test denoted that

Table 4 Ultrasound findings between the studied groups

Ultrasound findings	Group I (*N* = 31) [*n* (%)]	Group II (*N* = 31) [*n* (%)]	Group III (*N* = 31) [*n* (%)]	Test of significance	*P*-value
Liver					
Normal	14 (45.2)	9 (29.0)	31 (100.0)	χ^2 = 37.848*	<0.001*
Cirrhosis	8 (25.8)	15 (48.4)	0 (0.0)		
Fatty liver	9 (29.0)	7 (22.6)	0 (0.0)		
Pair-wise comparison[a]	I–II***, II–III***				
Right lobe (cm)					
Mean ± SD	12.81 ± 1.08	12.26 ± 1.0	12.45 ± 0.51	*F* = 2.979	0.056
Minimum–maximum	11.0–16.0	11.0–15.0	12.0–13.0		
PV (mm)					
Mean ± SD	13.35 ± 1.64	14.74 ± 1.18	12.48 ± 0.51	*F* = 27.669*	<0.001*
Minimum–maximum	11.0–17.0	12.0–17.0	12.0–13.0		
Pair-wise comparison[b]	I–II***, I–III*, II–III***				
Fibrosis					
Absent	31 (100.0)	0 (0.0)	31 (100.0)	$^{KW}\chi^2$ = 89.155*	<0.001*
Grade I	0 (0.0)	23 (74.2)	0 (0.0)		
Grade II	0 (0.0)	8 (25.8)	0 (0.0)		
Pair-wise comparison[b]	I–III***, II–III***				
GB					
Normal	28 (90.3)	27 (87.1)	31 (100.0)	χ^2 = 6.302	^{MC}P = 0.124
Cholecystitis	2 (6.5)	4 (12.9)	0 (0.0)		
Gall stones	1 (3.2)	0 (0.0)	0 (0.0)		
Spleen (cm)					
Mean ± SD	13.35 ± 1.18	15.90 ± 3.11	12.61 ± 0.50	*F* = 24.492*	<0.001*
Minimum–maximum	12.0–16.80	12.0–18.0	12.0–13.0		
Pair-wise comparison[b]	I–II***, II–III***				

F, *F*-test (analysis of variance); GB, gall bladder; group I, HCV; group II, HCV + schistosomiasis; group III, control; HCV, hepatitis C virus; KW, Kruskal–Wallis test; MC, Monte Carlo test; PV, portal vein. [a]Pair-wise comparison was made using the Monte Carlo test. [b]Pair-wise comparison was made using the post-hoc test (Scheffe). *Statistically significant at $P \leq 0.05$. **Statistically significant at $P \leq 0.01$. ***Statistically significant at $P \leq 0.001$.

there was a difference in the mean platelet count; it was greater in group I compared with group II, lower in group I compared with group III, and lower in group II compared with group III.

Liver functions

Table 6 shows the liver functions of the studied groups.

There was a significantly high statistical difference between the three studied groups with regard to ALT level. Pair-wise comparison test denoted that there was a difference in the mean ALT level; it was lower in group I compared with group II, greater in group I compared with group III, and greater in group II compared with group III.

There was a significantly high statistical difference between the three studied groups with regard to AST level. Pair-wise comparison test denoted that there was a difference in the mean AST level; it was lower in group I compared with group II, greater in group I compared with group III, and greater in group II compared with group III.

There was a significantly high statistical difference between the three studied groups with regard to albumin concentration. Pair-wise comparison test denoted that there was a difference in the mean albumin concentration; it was greater in group I compared with group II, lower in group I compared with group III, and lower in group II compared with group III (Fig. 3).

All participants had normal serum bilirubin and prothrombin activity.

Lipid profile

Table 7 shows the lipid profile of the studied groups.

Table 5 Comparison between the studied groups with regard to CBC

CBC parameters	Group I (*N* = 31)	Group II (*N* = 31)	Group III (*N* = 31)	*F*	*P*-value
RBCs (10^6/mm^3)					
Mean ± SD	4.70 ± 0.32	4.13 ± 0.23	5.25 ± 0.24	139.065*	<0.001*
Minimum–maximum	4.20–5.30	3.70–4.90	4.80–5.60		
Pair-wise comparison	I–II***, I–III***, II–III***				
Hb (g/dl)					
Mean ± SD	12.23 ± 0.50	11.01 ± 0.47	14.33 ± 0.55	334.771*	<0.001*
Minimum–maximum	11.30–13.20	10.30–11.90	13.0–15.50		
Pair-wise comparison	I–II***, I–III***, II–III***				
WBCs (10^3/mm^3)					
Mean ± SD	6.52 ± 0.85	6.23 ± 0.80	6.50 ± 0.89	1.117	0.332
Minimum–maximum	4.50–8.20	4.90–8.0	4.50–8.20		
Pair-wise comparison	I–II**, II–III*				
Platelets (10^3/mm^3)					
Mean ± SD	195.13 ± 33.39	127.58 ± 14.95	343.55 ± 37.16	417.410*	<0.001*
Minimum–maximum	124.0–284.0	105.0–155.0	250.0–398.0		
Pair-wise comparison	I–II***, I–III***, II–III***				

Pair-wise comparison was made using the post-hoc test (Scheffe). CBC, complete blood count; *F*, *F*-test (analysis of variance); group I, HCV; group II, HCV + schistosomiasis; group III, control; Hb, hemoglobin; HCV, hepatitis C virus; RBC, red blood cell; WBC, white blood cell. *Statistically significant at $P \leq 0.05$. **Statistically significant at $P \leq 0.01$. ***Statistically significant at $P \leq 0.001$.

Table 6 Comparison between the studied groups with regard to liver functions

Liver functions	Group I (*N* = 31)	Group II (*N* = 31)	Group III (*N* = 31)	F	*P*-value
ALT (IU/l)					
Mean ± SD	38.19 ± 2.90	44.48 ± 2.69	34.42 ± 2.35	113.381*	<0.001*
Minimum–maximum	34.0–44.0	41.0–49.0	31.0–39.0		
Pair-wise comparison	I–II***, I–III***, II–III***				
AST (IU/l)					
Mean ± SD	38.52 ± 4.07	45.71 ± 3.33	29.42 ± 2.35	186.748*	<0.001*
Minimum–maximum	32.0–49.0	34.0–49.0	26.0–34.0		
Pair-wise comparison	I–II***, I–III***, II–III***				
Albumin (g/dl)					
Mean ± SD	3.70 ± 0.25	3.12 ± 0.28	4.40 ± 0.14	238.190*	<0.001*
Minimum–maximum	3.20–4.20	2.40–3.60	4.10–4.60		
Pair-wise comparison	I–II***, I–III***, II–III***				

Pair-wise comparison was made using the post-hoc test (Scheffe). ALT, alanine transaminase; AST, aspartate transaminase; *F*, *F*-test (analysis of variance); group I, HCV; group II, HCV + schistosomiasis; group III, control; HCV, hepatitis C virus. *Statistically significant at $P \leq 0.05$. ***Statistically significant at $P \leq 0.001$.

There was no statistical difference between the three studied groups with regard to serum cholesterol and triglyceride concentration.

Leptin level

Table 8 shows the leptin profile of the studied groups.

There was a significant statistical difference between the three studied groups with regard to leptin level. Pair-wise comparison test denoted that there was a difference in the mean leptin level, being greater in group II compared with group III (Fig. 4).

Fat percentage

Table 9 shows fat percentage in the different studied groups.

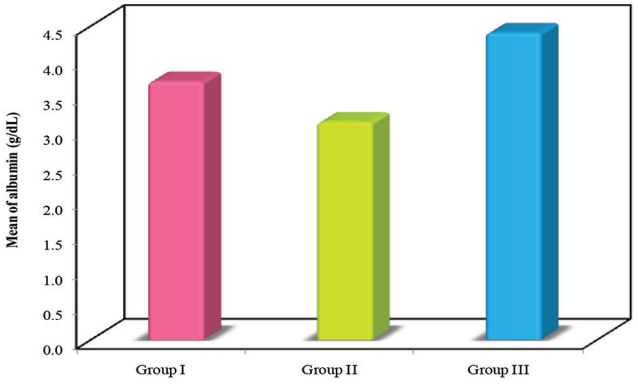
There was a significantly high statistical difference between the three studied groups with regard to fat percentage. The highest fat percentage was seen in controls and the lowest fat percentage was seen in group II. Pair-wise comparison test denoted that there was a difference in the fat percentage; it was greater in group I compared with group II, lower in group I compared with group III, and lower in group II compared with group III (Fig. 5).

Discussion

The present study aimed to evaluate the nutritional status of HCV-infected patients in association with schistosomal hepatic periportal fibrosis.

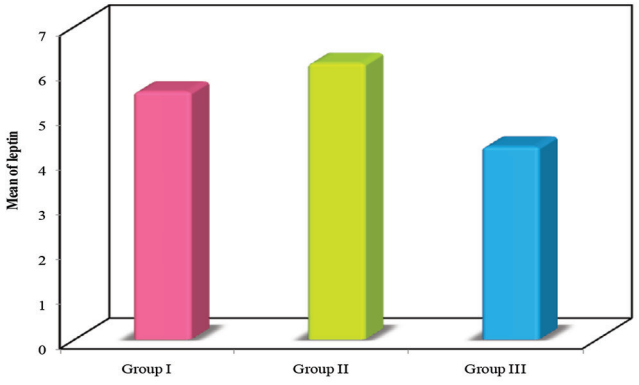
This study showed that the two studied groups (single infection and mixed infection) had malnutrition compared with the control group. The degree of malnutrition was more severe in group II, which had mixed schistosomiasis and HCV infections.

Figure 3



Comparison between the studied groups with regard to albumin.

Figure 4



Comparison between the studied groups with regard to leptin level.

Table 7 Comparison between the studied groups with regard to lipid profile

Lipid profile	Group I (*N* = 31)	Group II (*N* = 31)	Group III (*N* = 31)	*F*	*P*-value
Cholesterol (mg/dl)					
Mean ± SD	143.94 ± 31.04	143.42 ± 20.17	142.87 ± 31.95	0.011	0.989
Minimum–maximum	84.0–210.0	110.0–190.0	90.0–197.0		
Triglycerides (mg/dl)					
Mean ± SD	118.13 ± 31.92	118.42 ± 35.01	132.61 ± 42.12	1.586	0.210
Minimum–maximum	78.0–200.0	68.0–187.0	64.0–220.0		
Pair-wise comparison	II–III				

Pair-wise comparison was made using the post-hoc test (Scheffe). *F*, *F*-test (analysis of variance); group I, HCV; group II, HCV + schistosomiasis; group III, control; HCV, hepatitis C virus. *Statistically significant at $P \leq 0.05$. **Statistically significant at $P \leq 0.01$.

Table 8 Comparison between the studied groups with regard to leptin level

Leptin level	Group I (*N* = 31)	Group II (*N* = 31)	Group III (*N* = 31)	*F*	*P*-value
Leptin (ng/ml)					
Mean ± SD	5.54 ± 2.34	6.18 ± 1.88	4.31 ± 2.61	5.317*	0.007*
Minimum–maximum	2.90–13.0	3.0–13.0	0.50–11.30		
Pair-wise comparison	II–III**				

Pair-wise comparison was made using the post-hoc test (Scheffe). *F*, *F*-test (analysis of variance); group I, HCV; group II, HCV + schistosomiasis; group III, control; HCV, hepatitis C virus. *Statistically significant at $P \leq 0.05$. **Statistically significant at $P \leq 0.01$.

The chief reason for the malnutrition in these patients is poor oral intake, which may be due to a variety of causes. Vitamin A and or zinc deficiency may give rise to an altered sense of taste [17]. The dietary restrictions that are frequently advised to these patients, such as restriction of salt, protein, and fats, can discourage adequate oral intake by rendering food a bland taste. Weakness, fatigue, and encephalopathy may also contribute to decreased oral intake [18].

In addition, the loss of appetite may be related to the increased regulation of inflammation and of appetite mediators. Besides the hormonal influences and physical discomfort, the lack of interest in food may result from food restrictions and changes in taste [19].

Dietary limitations such as sodium restriction for the control of ascites, preoperative fast, and limitation of protein intake due to severe hepatic encephalopathy may reduce the variety of foods and many patients do not accept the recommended foods. Although changes in taste may be commonly attributed to micronutrient deficiencies, several investigators have questioned whether they might be a consequence of cirrhosis itself [19].

Malabsorption is another vital reason why patients with advanced hepatic disease become malnourished. A reduction in the bile-salt pool may lead to fat malabsorption [19], or bacterial overgrowth may result from impaired small-bowel motility [20]. Portal hypertension has also been named as a cause of malabsorption and protein loss from the gastrointestinal tract [21,22].

Malabsorption may also be caused by pancreatic insufficiency and cholestasis and may be related to drugs that cause diarrhea (lactulose, antibiotics, diuretics, and cholestyramine). Several mechanisms can lead to the malabsorption of nutrients, fats in particular, in cirrhotic patients. A complication that affects nutrient absorption is the portosystemic shunt. With the progression of cirrhosis, the nutrients bypass the liver through the portosystemic shunt without being processed metabolically [23].

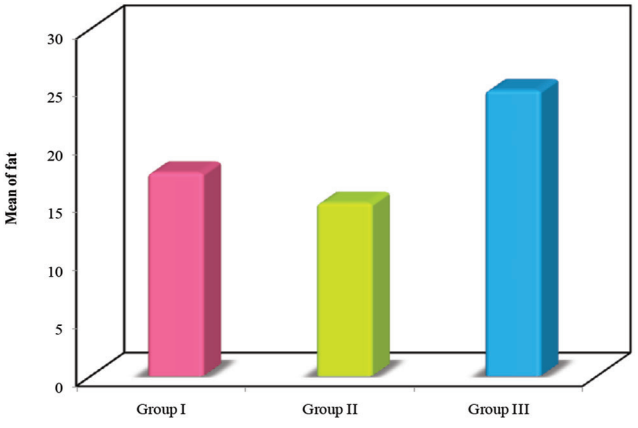
The functional integrity of the liver is essential for the utilization of nutrients. The liver influences the nutritional status by its production of bile acids and its role in the intermediate metabolism of proteins, carbohydrates, fats, and vitamins [24].

Among the metabolic disorders we may mention the following: hypermetabolism during complications such as infections, hemorrhage, decompensation, and ascites; increased protein catabolism due to inflammation and impaired hepatic synthesis; reduced glucose homeostasis due to hepatic insulin resistance caused by changes in gluconeogenesis, low glycogen stores, and impaired glycogenolysis; and increased lipolysis and lipid oxidation and proinflammatory cytokines (tumor necrosis factor α, interleukins, and leptin) [23].

Malnutrition may also be related to iatrogenic causes involved in investigative procedures, as well as to fasting periods, protein restriction during periods of encephalopathy, and to large volumes of paracentesis [23].

Energy expenditure is also known to contribute to the decline of nutritional status [23]. Although most cirrhotic patients have resting energy expenditure similar to predicted values, 15 to 30% of them are hypermetabolic. Hypermetabolism may be defined as an resting energy expenditure of 120% compared with predicted values [23].

Figure 5



Comparison between the studied groups with regard to fat percentage.

Table 9 Fat percentage of the different studied groups

Fat percent	Group I (*N* = 31)	Group II (*N* = 31)	Group III (*N* = 31)	*F*	*P*-value
Fat percentage					
Mean ± SD	17.56 ± 2.74	14.95 ± 2.91	24.69 ± 3.41	85.607*	<0.001*
Minimum–maximum	11.80–24.60	10.40–25.90	20.0–34.80		
Pair-wise comparison	I–II**, I–III***, II–III***				

Pair-wise comparison was made using the post-hoc test (Scheffe). *F*, *F*-test (analysis of variance); group I, HCV; group II, HCV + schistosomiasis; group III, control. *Statistically significant at $P \leq 0.05$. **Statistically significant at $P \leq 0.01$. ***Statistically significant at $P \leq 0.001$.

The nutritional assessment of these patients is a challenge and should be performed with caution, as changes inherent to the liver disease itself, such as edemas, ascites, and protein changes, impair this task, preventing the use of the more traditional parameters for nutritional assessment. The 2006 guidelines of the European Society of Enteral and Parenteral Nutrition recommend the use of subjective global assessment, anthropometric analysis, and hand-grip strength test to identify patients with cirrhosis who are at risk for malnutrition. Thus, subjective global assessment, anthropometric measurements, and the hand-grip strength test are more commonly used in routine nutritional assessment. However, there is no gold-standard method of easy application and low-cost, without subjective data and not influenced by the professional who performs it [23].

Albumin and lipids are poor nutritional markers, because they are typically reduced in patients with advanced hepatic disease and fluctuate during periods of inflammation [23].

Leptin is an adipokine that contributes to the pathogenesis of liver steatosis. In patients with chronic hepatitis C, higher serum leptin concentrations have been associated with the presence of steatosis [25].

Several studies have shown that circulating leptin levels are modestly elevated in patients with alcoholic cirrhosis, suggesting that leptin might be involved in the malnutrition of cirrhosis [25]. Although some studies have supported these findings, others have reported low serum leptin levels in posthepatitis cirrhotic patients [26]. In addition, nutritional status of cirrhotic cases represents a wide range in normal-to-severe malnutrition, connected with severity of the disease. It appears that relationship of serum leptin levels and nutritional status in posthepatitis cirrhosis has not been fully clarified yet [27].

In a recent study, Fernandes *et al*. [28] found that the BIA is the only way for nutritional assessment that is correlated with the severity of hepatic disease assessed using the Child–Pugh classification and for the classification of malnutrition in the population of cirrhotic patients.

Thus, the interest in comparing the BIA with other methods used for the nutritional assessment of cirrhotic patients is clearly justified, to obtain data about its performance as an indicator of the nutritional status of these patients. In addition, a precise, low-cost, and reproducible nutritional parameter could be included in the Child–Pugh and model for end-stage liver disease equations, contributing to a better prognosis for patients.

Conclusion

This study showed that the patients infected by HCV with and without schistosomal periportal fibrosis have malnutrition compared with healthy controls. The degree of malnutrition was more severe in patients having mixed schistosomal periportal fibrosis and HCV infections.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1 David AR. 5000 years of schistosomiasis in Egypt. Chungará (Arica) 2000; 32:133–135.

2 Sarvel AK, Oliveira AA, Silva AR, Lima AC, Katz N. Evaluation of a 25-year-program for the control of schistosomiasis mansoni in an endemic area in Brazil. PLoS Negl Trop Dis 2011; 5:e990.

3 Utzinger J, Raso G, Brooker S, de Savigny D, Tanner M, Ornbjerg N, *et al*. Schistosomiasis and neglected tropical diseases: towards integrated and sustainable control and a word of caution. Parasitology 2009; 136: 1859–1874.

4 Mohamed MK, Rakhaa M, Shoeir S, Saber M. Viral hepatitis C infection among Egyptians the magnitude of the problem: epidemiological and laboratory approach. J Egypt Public Health Assoc 1996; 71:79–111.

5 Krenitski J. Nutrition for patients with hepatic failure. Pract Gastroenterol 2003; 6:23–42.

6 Carvalho L, Parise ER. Evaluation of nutritional status of non hospitalized patients with liver cirrhosis. Arq Gastroenterol 2006; 43:269–274.

7 Charlton M. Branched-chain amino acid enriched supplements as therapy for liver disease. J Nutr 2006; 136(Suppl): 295S–298SS.

8 Ahima RS, Flier JS. Leptin. Ann Rev Physiol 2000; 62:413–437.

9 Simons JP, Schols AM, Westerterp KR, ten Velde GP, Wouters EF. The use of bioelectrical impedance analysis to predict total body water in patients with cancer cachexia. Am J Clin Nutr 1995; 61:741–745.

10 Kyle UG, Bosaeus I, de Lorenzo AD, Deurenberg P, Elia M, Gómez JM, *et al*. Bioelectrical impedance analysis – part I: review of principles and methods. Clin Nutr 2004; 23:1226–1243.

11 WHO. Ultrasound in schistosomiasis. A practical guide to the standardized use of ultrasonography for assessment of schistosomiasis related morbidity; 1996.

12 Knopp S, Speich B, Hattendorf J, Rinaldi L, Mohammed KA, Khamis IS, *et al*. Diagnostic accuracy of Kato-Katz and FLOTAC for assessing anthelmintic drug efficacy. PLoS Negl Trop Dis 2011 5:e1036.

13 Kinkel HF, Dittrich S, Bäumer B, Weitzel T. Evaluation of eight serological tests for diagnosis of imported schistosomiasis. Clin Vaccine Immunol 2012; 19:948–953.

14 Babiarczyk B, Turbiarz A. Body mass index in elderly people – do the refernce ranges matter? Prog Health Sci 2012; 2:58.

15 Peltz G, Aguirre MT, Sanderson M, Fadden MK. The role of fat mass index in determining obesity. Am J Hum Biol 2010; 22:639–647.

16 Gallagher D, Heymsfield SB, Heo M, Jebb SA, Murgatroyd PR, Sakamoto Y. Healthy percentage body fat ranges: an approach for developing guidelines based on body mass index. Am J Clin Nutr 2000; 72:694–701.

17 Garrett-Laster M, Russell RM, Jacques PF. Impairment of taste and olfaction in patients with cirrhosis: the role of vitamin A. Hum Nutr Clin Nutr 1984; 38:203–214.

18 McCullough AJ, Bugianesi E. Protein-calorie malnutrition and the etiology of cirrhosis. Am J Gastroenterol 1997; 92:734–738.

19 Vlahcevic ZR, Buhac I, Farrar JT, Bell CC Jr, Swell L. Bile acid metabolism in patients with cirrhosis. I. Kinetic aspects of cholic acid metabolism. Gastroenterology 1971; 60:491–498.

20 Gunnarsdottir SA, Sadik R, Shev S, Simrén M, Sjövall H, Stotzer PO, *et al*. Small intestinal motility disturbances and bacterial overgrowth in patients

with liver cirrhosis and portal hypertension. Am J Gastroenterol 2003; 98:1362–1370.

21 Romiti A, Merli M, Martorano M, Parrilli G, Martino F, Riggio O, *et al*. Malabsorption and nutritional abnormalities in patients with liver cirrhosis. Ital J Gastroenterol 1990; 22:118–123.

22 Conn HO. Is protein-losing enteropathy a significant complication of portal hypertension?. Am J Gastroenterol 1998; 93:127–128.

23 Cheung K, Lee SS, Raman M. Prevalence and mechanisms of malnutrition in patients with advanced liver disease, and nutrition management strategies. Clin Gastroenterol Hepatol 2012; 10:117–125.

24 Friedman S, Schiano T. Cirrhosis and its sequelae. In: Goldman L, Ausiello D, eds. *Cecil textbook of medicine*. 22nd ed Philadelphia, PA: Saunders; 2004. 936–944.

25 Ockenga J, Bischoff SC, Tillmann HL, Rifai K, Widjaja A, Böker KH, *et al*. Elevated bound leptin correlates with energy expenditure in cirrhotics. Gastroenterology 2000; 119:1656–1662.

26 McCullough AJ, Bugianesi E, Marchesini G, Kalhan SC. Genderdependent alterations in serum leptin in alcoholic cirrhosis. Gastroenterology 1998; 115:947–953.

27 Greco AV, Mingrone G, Favuzzi A, Capristo E, Gniuli D, Addolorato G, *et al*. Serum leptin levels in post-hepatitis liver cirrhosis. J Hepatol 2000; 33:38–42.

28 Fernandes SA, Bassani L, Nunes FF, Aydos MED, Alves AV, Marroni CA. Nutritional assessment in patients with cirrhosis. Arq Gastroenterol 2012; 49:19–27.