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MicroRNAs Correlation with Liver Function and Lipid Profiles Among Hepatitis C Virus (HCV) Patients as Diagnostic Biomarkers

Lubna Hussein Murshed [1], Jaleel Samanje [1], Dr. Athraa Zaidan Hassan [1], Dr. Safa. A. AL-Waisy [2],

[1] College of Health and Medical Techniques, Middle Technical University, Baghdad, Iraq.
[2] GIT teaching hospital, Medical city Health directorate, Iraqi Ministry of health, Baghdad, Iraq.

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Correspondence: Lubna Hussein Murshed
Email: edc4005@mtu.edu.iq

Website: https://djm.uodiyala.edu.iq/index.php/djm

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Abstract

Background: MicroRNAs have been concerned in modulating multiple stages of HCV life cycles and specific miRNAs have been identified to be deregulated during HCV infection and serve as essential mediators for the antiviral treatment.

Objectives: To understand the role of circulating serum microRNAs (miR-21-5p and miR-196-5p) in correlation with liver function tests and lipid profiles as diagnostic markers in patients with different stages of hepatitis C virus (HCV).

Patients and Methods: 150 cases—100 with the Hepatitis C virus and 50 healthy as a control group—are involved. The patients' and the control group's serum levels of miR-21-5p and miR-196-5p were assessed using quantitative real-time PCR (qRT-PCR), and the biochemical tests were measured using a standard automatic biochemistry analyzer (Thermo Fisher 240 V Indiko Plus Clinical Chemistry Analyzer, Kerala, India).

Results: Serum miR-21-5p is upregulated in patients with acute and chronic HCV compared with the control group, while serum miR-196-5p is upregulated in acute HCV, HCV induced liver cirrhosis and sustained virologic response patients compared to the control group. The maximum serum total cholesterol (TC), TG, LDL and VLDL levels were in SVR and the minimum levels were in HCV-LC, the highest LFTs levels were in the AHC group. MiR-196-5p was negatively correlated with TG and VLDL in AHC and HCV-LC. The ROC curve showed the highest sensitivity and specificity for miR-21-5p in the AHC and CHC groups, while the highest sensitivity and specificity for miR-196-5p was in the AHC, HCV-LC, and SVR groups.

Conclusion: Increased serum miR-21-5p and miR-196-5p expression in HCV patients implicated in lipid dysregulation within hepatocytes and monitoring this correlation might be used as biomarker of disease progression and liver dysfunction.

Keywords: HCV, miR-21-5p, miR-196-5p, Circulating, Biomarker.

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Introduction

Hepatitis C virus (HCV) belongs to the family Flaviviridae of the genus Hepacivirus which is a positive-sense RNA virus and primarily affects the liver leading to acute or chronic liver inflammation and potentially resulting in chronic liver complications such as fibrosis, cirrhosis, and development of hepatocellular carcinoma (HCC) (1). Since, HCV is a non-cytopathic virus, immune deregulation is the primary pathogenesis of these HCV associated liver diseases (2). Although, chronic hepatitis, liver cirrhosis (LC) and hepatocellular carcinoma can result from multiple etiologies such as alcohol consumption, non-alcoholic fatty liver disease and chronic viral infections including HCV and HBV (3), but HCV-related cirrhosis and liver cancer were the cause of death of roughly 242,000 people in 2022, with about one million new infections occurring annually (4). The serological tests used to diagnose HCV do not identify the stage of the infection—acute, chronic, or resolved—and cirrhosis is frequently undetected. In addition, liver biopsy is linked to complications like pain and bleeding. Therefore, in order to accurately determine the stage of liver disease associated with HCV, new non-invasive biomarkers are required. MicroRNAs (miRNAs), which are a type of remarkably preserved small non-coding RNAs with 19–25 nucleotides in length that can bind partially or completely to target messenger RNAs and regulate gene expression at post-transcription level by mechanisms like mRNA degradation or translation inhibition (5), are identified as important in pathogenesis of HCV infection and some deregulated circulating miRNAs have been considered as non-invasive biomarkers for the detection of HCV infection and/or HCV-related diseases (6). In this work, miRNAs-21 and 196 were chosen to be studied. MiRNA-196 was chosen because HCV infection upregulates the expression of miRNA-196b, which can inhibit HCV through either directly targeting virus protein (NS5A) or indirectly targeting host genes BTB domain and CNC homolog 1 (BACH1) and heme oxygenase 1 gene (HMOX1) (6). However, its application as a biomarker may be challenging, though, due to contradictory findings regarding whether it is upregulated or downregulated during HCV infection (7). MiRNA-21 which is a type of miRNA found in the liver was chosen for its implication in diverse biological processes, including inflammation, fibrosis, and cancer (8). HCV infection includes all these biological processes in its different stages; therefore, there may be a link between HCV infection and miRNA-21 which can be targeted in the future to reduce the progression of HCV to cirrhosis and subsequently hepatocellular carcinoma. Liver function tests, or LFTs, are essential in the management of Hepatitis C (HCV) and are used to identify liver damage, evaluate the severity of the disease, and track the efficacy of treatment. These blood tests quantify the blood's levels of proteins and enzymes, which, when abnormal, can reveal the stage of disease (inflammation, fibrosis, cirrhosis). However, normal liver function tests do not usually rule out liver damage (9). Infection with the hepatitis C virus significantly affects lipid metabolism, leading to alterations in serum lipid profiles according to disease progression and treatment outcomes. HCV has been revealed to increase lipid biosynthesis and impair lipid catabolism, causing hypocholesterolemia and intracellular accumulation of lipids (10). Direct-acting antivirals (DAAs) also have been suggested to affect serum lipid profiles especially low-density lipoprotein (LDL), high-density lipoprotein (HDL) and triglyceride (11). This study aimed to estimate the role of microRNAs (21-5p and 196-5p) and their correlation with liver function tests and lipid profiles as diagnostic biomarkers in different stages of HCV infection. compositions of the products they consumed, highlighting the

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Patients and Methods

Patient enrollment: In this prospective study which registered during the period from May 2024 to February 2025, a total of 150 blood samples were collected from five groups of participants: Group I included 25 cases of acute hepatitis C whose ages ranged between 5 and 41 years. Group II comprised 25 patients of chronic hepatitis C with ages ranging between 12 and 63 years. Group III included 25 cases of (HCV induced liver cirrhosis) whose ages ranged between 25 and 81 years and Group IV comprised 25 treated HCV patients known as Sustained Virologic Response (SVR), while Group V (control group) included 50 obviously healthy individuals with ages between 5 and 81 years who were negative for viral screen by ELISA. Inclusion criteria were based on a positive enzyme-linked immunosorbent assay (ELISA) test and a positive PCR viral load test for HCV in groups I,II and III, but a negative PCR viral load test for HCV in group IV (SVR group). The exclusion criteria included the absence of other liver diseases, autoimmune, metabolic disorders, co-infection with hepatitis B virus and/or human immunodeficiency virus, malignancies and alcohol abuse, pregnant women were also excluded.

Serum collection and storage: Venous blood samples were collected from each individual enrolled in this study and placed into anticoagulant-free gel tubes, then centrifuged at 4000g for 10 min, and the supernatant serum was collected. Afterwards, serum was subdivided into two aliquots; 0.5ml was placed in a tube containing 0.5ml TRIzol™ reagent for RNA saving in intact form and kept at -20 ºC for the time of molecular analysis. The second one was placed in Eppendorf tubes and frozen at -20 ºC until used for liver function tests and lipid profile investigations.

Molecular detection of miRNAs: 56 sample were selected for molecular detection of miR-196-5p and miR-21-5p which was carried out according to (GoScript Reverse Transcription System, Promega, USA) beginning with RNA extraction followed by two step reverse transcription (Complementary DNA synthesis followed by Real-time quantitative PCR for detection the expression levels of mature miR-196-5p and miR-21-5p).Briefly, RT-qPCR amplification was performed with gene specific forward primer and a reverse primer (from Macrogen Company, Korea) along with a probe in a Magnetic induction cycler (Mic) qPCR Cycler (Bio Molecular System, Australia), preceded by first strand cDNA synthesis with 10 ng RNA and miRNA-196-5p/ miRNA-21-5p specific, RT-primer, a control endogenous miRNA (RNU43). The primers used in the reverse transcription step and PCR were designed as follows (Table 1).

Biochemical tests: Serum levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), albumin, total serum bilirubin (TSB), gamma glutamyl transferase (γGT), alkaline phosphatase (ALP), total Cholesterol (TC), high density cholesterol (HDL), low density cholesterol (LDL), very low density cholesterol (VLDL) and triglycerides (TG) in patients with HCV and healthy controls were detected using a standard automatic biochemistry analyzer (Thermo Fisher 240 V Indiko Plus Clinical Chemistry Analyzer, Kerala, India).

Statistical Analysis

Calculations were performed using Statistical Packages for Social Sciences- version 26, software (SPSS-26) from Chicago, USA. The normality test was used to determine whether the variables were normally distributed or not. The significance of the difference of normally distributed continuous quantitative data was tested with ANOVA test, while the significance of the difference between non-normally distributed continuous quantitative data was

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tested using the Kruskal-Wallis H test for differences between more than two independent means or the Mann-Whitney U test for differences between two independent means. When the P value was equal to or less than 0.05, statistical significance was taken into account.

The correlation between serum microRNAs and biochemical tests was computed using Spearman's rank correlation. The sensitivity and specificity of microRNAs to be used as a disease diagnostic or screening tool was assessed by the Receiver Operating Characteristic "ROC" curve.

Table 1. The primers used in the reverse transcription step and PCR.

Primers Name	Sequence 5́-3́	Annealing Temp.
miR-21-5p-RT	GTTGGCTCTGGTGCAGGGTCCGAGGTATTCG CACCAGAGCCAAC TCAACA	60 ºC
miR-21-5p-F	GTTTGGTAGCTTATCAGACTGA	
miR-196-5p-RT	GTTGGCTCTGGTGCAGGGTCCGAGGTATTCG CACCAGAGCCAAC CCCAAC	
miR-196-5p-F	GTTGGGTAGGTAGTTTCATGTT	
RNU43-RT	GTTGGCTCTGGTGCAGGGTCCGAGGTATTCG CACCAGAGCCAACAATCAG	55 ºC
RNU43-F	GTGAACTTATTGACGGGCG	
Universal Reverse	GTGCAGGGTCCGAGGT	
Relative miRNA expression was calculated using the ΔΔCT method; it is given by the equations: ΔCT = CT gene - CT House Keeping gene ΔΔCT = ΔCT one patient – Average ΔCT Controls Relative Folding Change (relative gene expression) =2-ΔΔCT Folding change difference was calculated for each group by dividing the mean expression level for each group by the mean expression level of the control group.		

Results

Distribution of the studied groups according to age (years): HCV patients and the control group were age-matched (36.55±18.8 and 35.7 ±18.9 years) respectively, and there was no significant difference between these two groups (P= 0.79), while the difference in mean age between each HCV group and the control group was highly significant at (p<0.01) levels [Table 2].

Comparison the serum levels of liver function tests: The result [Table 3] of the comparison of mean serum levels of LFTs among study groups showed a highly significant difference in ALT, AST and GGT levels between the AHC group compared with the CHC, LC, SVR & healthy groups and between the CHC compared to liver cirrhosis (HCV-LC) groups (except for ALT in CHC vs. HCV-LC which was significant at 0.05 level), and HCV-LC compared to the SVR groups at p-value <0. 001, but only the AST & GGT was highly statically significant between the CHC compared to the HC groups and HCV-LC vs. HC group, and ALT with GGT in the SVR compared to the HC group. In addition, there were highly statistical differences in ALP between AHC compared to each group, and highly statistical differences in TSB between AHC groups compared to SVR & HC groups respectively, but it was significant at 0.05 levels between HCV-LC vs. HC groups. Serum albumin showed highly statistical differences between CHC and HCV-LC groups respectively compared to HC group and AHC compared to HCV-LC group, and significant difference in AHC group compared to CHC group and HCV-LC vs. SVR.

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Table 2. Distribution of study groups according to age in years.

Variables		Patients				Healthy control	P-value
		AHC	CHC	HCV-LC	SVR		
Age (year)	Range	5 - 41	12 - 63	25 - 81	17 - 72	5 - 81	0.0001**
	Mean ± SD	14.6 ±10.5	38.8 ±11.8	54.4 ±14.5	38.9 ±12.5	35.7 ±18.9	
	Mean ±SD	36.55±18.8 (Range= 5-81)				35.7±18.9	0.79
	Total No.	100				50	
** Highly significant							

Table 3. Comparison of serum levels of liver function tests.

Groups	Parameters in Mean ± SD					
	ALT (U/L)	AST (U/L)	GGT (IU/l)	TSB (mg/dl)	ALP (U/L)	Alb. (g/dl)
AHC	96.7±33	104±62	85.9±68.7	1.3±0.4	343.3±36	4.4±0.6
CHC	14.8±8.2	24.3±16.2	17±8.5	1.1±0.3	197.4±32.4	3.9±0.8
HCV-LC	28±18.9	41.2±21.6	28.7±9.3	0.9±0.2	183.4±26.7	3.7±0.7
SVR	12.7±4.8	17.4±6.8	16.7±5.9	0.7±0.1	142.3±11.6	4.3±0.9
HC	15.7±4.6	15.3±5.7	10±2.6	0.6±0.03	147.7±11.7	4.4±0.5
P-value	0.0001**	0.0001**	0.0001**	0.010**	0.0001**	0.001**
AHC vs. CHC	0.0001**	0.0001**	0.0001**	0.296	0.001**	0.018*
AHC vs. HCV-LC	0.0001**	0.0001**	0.0001**	0.265	0.0001**	0.001**
AHC vs. SVR	0.0001**	0.0001**	0.0001**	0.010**	0.0001**	0.281
AHC vs. HC	0.0001**	0.0001**	0.0001**	0.001**	0.0001**	0.902
CHC vs. HCV-LC	0.044*	0.007**	0.0001**	0.830	0.720	0.332
CHC vs. SVR	0.264	0.113	0.541	0.283	0.594	0.332
CHC vs. HC	0.481	0.010*	0.0001**	0.082	0.613	0.010 **
HCV-LC vs. SVR	0.008**	0.0001**	0.0001**	0.215	0.299	0.040*
HCV-LC vs. HC	0.105	0.0001**	0.0001**	0.029*	0.233	0.0001**
SVR vs. HC	0.006**	0.198	0.0001**	0.563	0.942	0.136
** = Highly significant, *= Significant.						

Comparison serum levels of lipid profile tests:

The results which were listed in table (4) showed that the maximum mean serum total cholesterol (TC), TG, LDL and VLDL levels were in SVR (271.5, 169.5, 200.9, and 33.9 mg/dl) respectively, and the minimum levels were in HCV-LC (TC=155.2, TG=136.1, LDL =97.5, VLDL=27.7 mg/dl) followed by CHC (TC=158.8, TG=136.8, LDL =105.6, VLDL=27.2 mg/dl) groups, while the maximum serum HDL level was in the control group (41.1mg/dl) followed by SVR group (36.5 mg/dl) with minimum levels in CHC group (24.4 mg/dl). Moreover, serum concentration of HDL-C showed a highly significant difference between the studied groups (P value <0.01). Also, observed a highly significant difference in the mean level of HDL-C when comparing AHC, CHC, HCV-LC and SVR groups respectively to HC, but a significant difference at P<0.05 levels when comparing AHC vs. CHC groups. The current study also showed a highly significant difference in LDL cholesterol mean levels between all groups and between the (AHC vs. SVR), (CHC vs. SVR), (LC vs. SVR) and (SVR vs. HC) at p-value < 0.01. While there were significant statistical differences in VLDL cholesterol at p-value ≤ 0.05 between the (AHC vs. HC), (CHC vs. HC) and (LC vs. HC) groups, this difference was not statically significant when comparing between all groups.

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Table 4. Serum levels of Lipid profile tests.

Groups	Parameters (by mg/dl) in Mean ± SD				
	TC	TG	HDL-C	LDL-C	VLDL-C
AHC	163.6±70.6	149.9±90.4	29.96±11.8	103.7±66.9	30±18.1
CHC	158.8±57.1	136.1±64.6	24.4±10.97	105.6±56.9	27.2±12.9
HCV-LC	155.2±69.3	136.8±51.5	30.2±8.01	97.5±68.8	27.7±10.3
SVR	271.5±91.7	169.5±67.4	36.5±15.4	200.9±90.5	33.9±13.5
HC	196.8±49.1	165.4±44.5	41.1±8.2	122.5±48.1	33.1±8.9
P-value comparing all	0.0001**	0.041*	0.0001**	0.0001**	0.051
AHC v CHC	0.839	0.946	0.036*	0.946	0.930
AHC vs. LC	0.648	0.793	0.900	0.567	0.778
AHC vs. SVR	0.0001**	0.065	0.438	0.0001**	0.071
AHC vs. HC	0.009**	0.020*	0.0001*	0.076	0.020*
CHC vs. LC	0.969	0.869	0.009**	0.648	0.801
CHC vs. SVR	0.0001**	0.133	0.001**	0.0001**	0.133
CHC vs. HC	0.004**	0.043 *	0.0001**	0.078	0.043*
LC vs.SVR	0.0001**	0.128	0.290	0.0001**	0.157
LC vs. HC	0.007**	0.028*	0.000**	0.037*	0.044*
SVR vs. HC	0.001**	0.719	0.007**	0.0001**	0.723
** = Highly significant, *= Significant.					

Comparison serum expression levels of miRNA-21-5p: Analysis of the mean fold change in the expression level of miRNA-21-5p in patients' serum in comparison to the healthy control group showed that miRNA-21-5p displayed a significant fold increase in expression in the HCV groups. [Table 5] shows a significant fold change increase in miRNA-21-5p expression between the (AHC vs. HCV-LC) (P=0.004), (AHC, CHC vs. control) (P=0.002, P < 0.01 respectively) and (AHC& CHC vs. SVR) groups, (P = 0.002 & < 0.01) respectively.

Table 5. Serum expression levels of miRNA-21-5p in the studied groups.

Parameter		Groups (No.)				
		AHC	CHC	HCV-LC	SVR	Control
Folding change difference (Mean ± SE)		131.196 ±63.44	38.99 ±11.2	4.102 ±3.26	2.144 ±0.7	1.00 ±0.27
Mean Rank		36.14	32.92	12.38	15.3	12.86
P-Value	AHC vs. CHC	0.257				
	AHC vs. HCV-LC	0.004**				
	AHC vs. SVR	0.002**				
	AHC vs. Control	0.002**				
	CHC vs. HCV-LC	0.001**				
	CHC vs. SVR	< 0.01**				
	CHC vs. Control	< 0.01**				
	HCV-LC vs. SVR	0.374				
	HCV-LC vs. Control	0.643				
	SVR vs. Control	0.625				
	P-value comparing all	< 0.01**				
** = Highly significant.						

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Comparison serum levels of miRNA-196-5p: Statistical analysis showed highly significant differences in the mean values of miRNA-196-5p in serum between the studied groups (P value =0.008) and also observed a highly significant fold change difference in miRNA-196-5p expression between the (CHC vs. SVR) group (P-value = 0.009) and significant fold change differences between the (AHC vs. control), (CHC vs. HCV-LC), (HCV-LC vs. control) & (SVR vs. control) (P = 0.023, P= 0.042, P= 0.013 & P= 0.011 respectively) [Table 6].

Table 6. MiRNA-196-5p serum levels.

Parameter		Groups (No.)				
		AHC	CHC	HCV-LC	SVR	Control
Folding change diff. (Mean ± SE)		25.75 ±11.5	3.33 ±1.15	15.36 ±5.8	24.24 ±8.2	1.00 ±0.39
Mean Rank		29.22	18.62	29.33	32.7	11.43
P-Value	AHC vs. CHC	0.102				
	AHC vs. HCV-LC	0.825				
	AHC vs. SVR	0.624				
	AHC vs. Control	0.023*				
	CHC vs. HCV-LC	0.042*				
	CHC vs. SVR	0.009**				
	CHC vs. Control	0.104				
	HCV-LC vs. SVR	0.514				
	HCV-LC vs. Control	0.013*				
	SVR vs. Control	0.011*				
	P-value comparing all	0.008**				
** = Highly significant, *= Significant.						

The estimation of spearman's rank correlations among studied parameters in the HCV patients' groups: The results of Spearman's Rank Correlations showed significant negative moderate correlation between miRNA-196-5p and age at p< 0.05 levels in the sustained virologic response group (SVR), and significant negative moderate correlation between miRNA-196-5p and serum Triglycerides (TG) and VLDL levels respectively in acute HCV group and with serum TG in HCV-LC group as seen in [Table 7]. MiR-21-5p showed no significant correlation between any of the studied parameters in all included groups.

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Table 7. Spearman correlation between the Levels of Biochemical tests with the expression levels of miRNA-196-5p and miRNA-21-5p in HCV Patients.

Biochemical tests		miRNA-196-5p				miRNA-21-5p			
		AHC (N=11)	CHC (N=13)	HCV-LC (N=10)	SVR (N=10)	AHC (N=11)	CHC (N=13)	HCV-LC (N=10)	SVR (N=10)
Age (years)	R	- 0.337	- 0.306	- 0.188	-0.722*	- 0.391	0.044	0.316	0.472
	P	0.311	0.334	0.602	0.018	0.235	0.893	0.374	0.168
ALT (U/L)	R	-0.200	0.306	0.261	-0.140	0.273	0.111	-0.377	-0.547
	P	0.555	0.334	0.466	0.699	0.417	0.718	0.283	0.101
AST (U/L)	R	0.492	0.091	0.527	-0.243	0.064	0.091	-0.588	-0.518
	P	0.124	0.779	0.117	0.498	0.852	0.768	0.074	0.125
GGT (IU/L)	R	0.064	-0.509	-0.042	0.116	0.296	-0.133	0.042	0.280
	P	0.852	0.090	0.907	0.751	0.377	0.666	0.907	0.432
TSB (mg/dl)	R	-0.207	0.208	0.140	-0.399	0.277	0.230	-0.055	0.271
	P	0.541	0.516	0.700	0.254	0.410	0.449	0.881	0.449
ALP (U/L)	R	0.109	-0.545	0.273	0.491	0.364	-0.036	-0.115	-0.201
	P	0.750	0.067	0.446	0.150	0.272	0.908	0.751	0.578
Alb. (g/dl)	R	0.182	-0.473	-0.321	0.103	-0.255	-0.017	0.285	0.237
	P	0.593	0.121	0.365	0.777	0.450	0.957	0.425	0.449
TC (mg/dl)	R	0.018	-0.210	-0.018	-0.442	-0.282	0.174	0.140	0.590
	P	0.958	0.513	0.960	0.200	0.401	0.571	0.700	0.073
TG (mg/dl)	R	-0.655*	-0.070	-0.745*	-0.309	0.409	-0.383	-0.018	-0.188
	P	0.029	0.829	0.013	0.385	0.212	0.197	0.960	0.602
HDL (mg/dl)	R	-0.173	-0.420	0.539	0.248	-0.145	-0.185	0.442	0.073
	P	0.612	0.175	0.108	0.489	0.670	0.546	0.200	0.841
LDL (mg/dl)	R	0.291	-0.112	-0.200	-0.600	-0.309	0.377	0.042	0.614
	P	0.385	0.729	0.580	0.067	0.355	0.204	0.907	0.059
VLDL (mg/dl)	R	-0.655*	-0.070	-0.564	-0.309	0.409	-0.383	-0.081	-0.188
	P	0.029	0.829	0.090	0.385	0.212	0.197	0.960	0.602

* Significant at 0.05 levels.

Estimation of ROC curve analysis of microRNAs among studied groups:

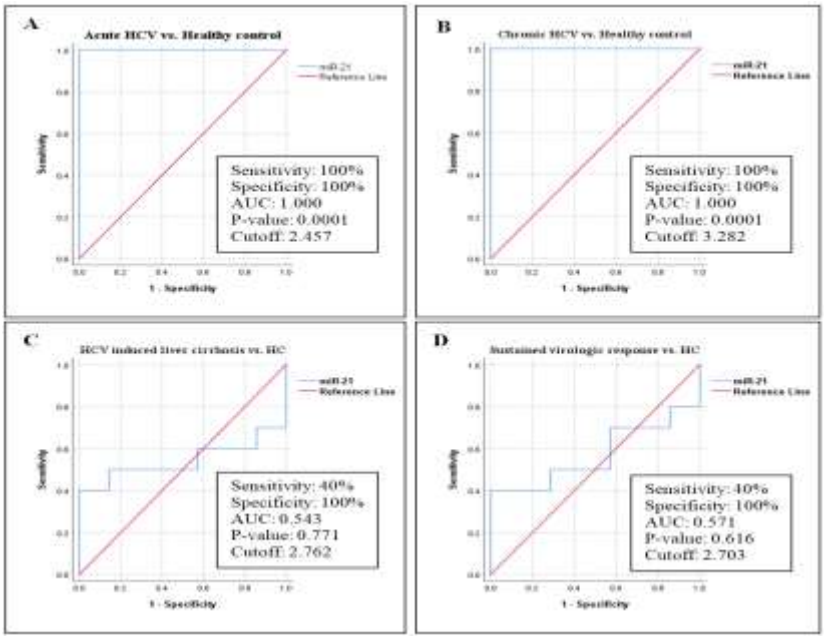

Figure 1. ROC curve analysis of serum miR-21-5p level in patients compared to healthy control group.

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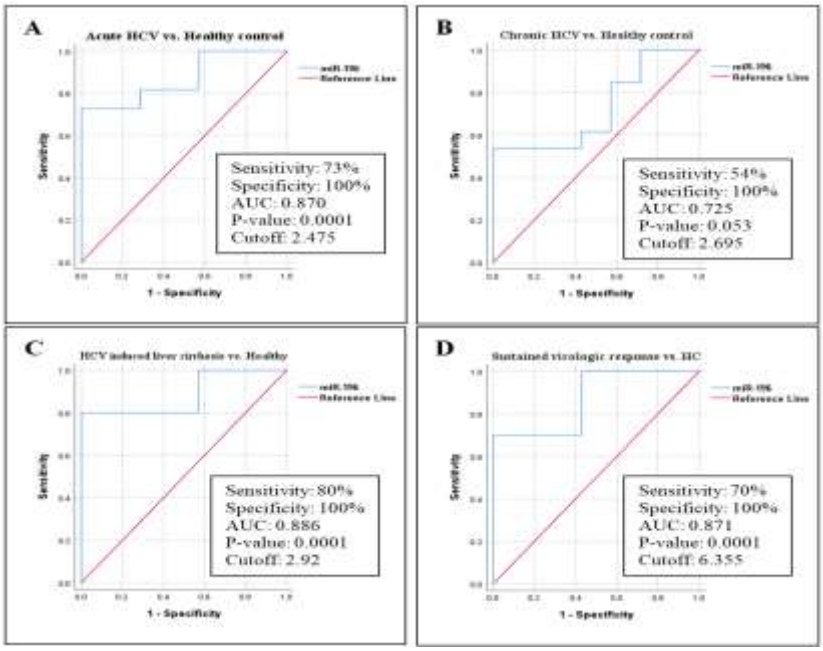

Figure 2. ROC curve analysis of serum miR-196-5p level in patients compared to healthy control group.

Discussion

There was a highly significant difference between the mean ages of all five included groups (P=0.0001), this indicates that the age of the sample had a high effect on the relationships between the studied parameters. These results were consistent with a previous study that showed the mean age in HCV-induced liver cirrhosis patients was significantly higher than the mean age of the chronic hepatitis C virus group (P=0.01) (12). However, the observation of this study was incompatible with a study that showed the studied groups were age-matched (P > 0.05) (13). This may be due to the fact that this age group has been exposed to more accidents, occupational risks, blood transfusions, and surgeries than younger groups of patients. The results of our study indicate that HCV is a slowly progressing disease and doesn't cause morbidity for many years. In addition, complications from HCV increase with increased age especially for those who remain undiagnosed. The response to treatment is higher at a younger age with a mean age of 38.9 years in this study, especially for those who are diagnosed early. The results of this study revealed that the higher abnormal levels of liver function tests (ALT, AST, GGT, TSB and ALP) were in the acute HCV group, and these elevated levels turned to normal levels in subsequent groups except for AST level in HCV –LC group which remained above the normal value but not as high as AHCV group. The elevation of liver function tests in acute HCV patients can be attributed to the ongoing damage of the liver tissue due to immune-mediated cell damage, as HCV is not a cytopathic virus, leading to inflammation and cell death due to apoptosis or necrosis (14, 2). About 30% of patients with chronic HCV can present with normal liver function tests due to a weaker immune response resulting in minimal liver injury, or some viral genotypes and variants associated with slow progressing disease and minimal liver damage. Also host genetic factors may affect the degree of liver damage and the release of liver enzymes; liver enzymes especially ALT and AST are not

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liver specific and may rise in the presence of substantial liver injury. However, normal liver function tests in chronic HCV patients do not rule out liver damage and imaging tests may show cirrhosis or fibrosis in these patients. In addition, chronic HCV with normal liver function tests may still progress to severe liver disease and cancer (15). The mean age of patients in AHC group was (14.6 years) as illustrated in [Table 2], this explains the higher ALP level in AHC group as ALP is normally higher during childhood and adolescence due to bone growth and this may reflect the origin of ALP in younger AHC patients may be the bone and not the liver. Results of serum lipid profile estimation showed that the maximum serum total cholesterol, TG, LDL and VLDL levels were in SVR and minimum levels in HCV-LC and CHC groups, while maximum serum HDL level was in control group followed by SVR group with minimum levels in CHC group. These results were in consistent with a study by Peschel et al., (16) who reported a significant increase in serum total cholesterol, LDL, VLDL, HDL and triglycerides. However, our results disagreed with a study by AbdEl-maksoud et al., (17) who found that serum levels of total cholesterol, TG, LDL and VLDL significantly decreased in HCV patients who achieved SVR, but it agreed with this study in increasing serum HDL cholesterol in these patients. HCV circulates in the blood as lipoviral particles by association with LDL and VLDL evading immune system and facilitating its entry to hepatocytes by binding to LDL receptors in hepatocytes and is responsible for decreased serum lipids because it inhibits the protein (microsomal triglycerides transfer) which is responsible for VLDL assembly and secretion, HCV also uses lipid metabolism pathways in the liver to facilitate its replication and secretion (18). Another cause of decreased serum lipids during HCV infection is increased triglycerides accumulation in the liver and reducing its secretion leading to hepatic steatosis by upregulation of SREBP-1c (a lipid uptake gene) and downregulation of lipids clearance by beta-oxidation process (19). In addition, increased expression of LDL receptor in hepatocytes by HCV leads to increased LDL uptake by hepatocytes, thus contributing to reduced circulating LDL cholesterol (20). Finally, chronic inflammation resulted from HCV infection induces cytokine secretion like (TNF-α and IL-6), these cytokines impair lipoprotein synthesis and lipid transport (21). The decreased serum HDL level during HCV infection is mostly due to altered HDL-cholesterol synthesis and catabolism (22). The mechanism of increased serum lipid profiles after achieving sustained virologic response in HCV patients is still unknown but evidence suggested that it may belong to the lack of virus direct interaction with host's lipid metabolism and most of these patients may have a metabolic syndrome especially those who had liver fibrosis (23). Data obtained in this study showed that the mean fold change difference in the expression level of miRNA-21-5p is significantly increased only in the acute and chronic hepatitis C groups when compared to the liver cirrhosis, SVR and healthy control groups. These findings correspond to the result of Manzoor et al., who reported that the mean expression level of miRNA-21 was significantly higher in patients with chronic HCV infection, while successful treatment of HCV decreases the expression of miR-21 (24). However, this study was incompatible with our study in that miR-21 upregulated with disease progression toward liver cirrhosis and HCC, while our study showed lower serum mean miR-21 expression level in HCV induced liver cirrhosis than CHC group. Also, a study by Hetta et al., (12) agreed with our study in that serum miR-21 significantly elevated in chronic hepatitis C compared to healthy control, but disagreed with our result in that serum miR-21 was higher in complicated HCV with cirrhosis

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than chronic hepatitis C group. However, there is no study revealing the role and expression mode of serum miR-21 in acute phase of hepatitis C virus. The decreased levels of circulating miR-21 in patients with liver cirrhosis may be due to reduced release from hepatocyte and it indicates that in these patients, the serum miRNA-21 level might be a marker for hepatic functional capacity, whereas at earlier stages of liver disease, the serum miRNA-21 level is mainly an indicator of neuroinflammatory activity and cell death in the liver. As release from damaged hepatocytes might be the major source of hepatocyte-derived miRNAs (25). It is conceivable that in cirrhotic patients who lost a big proportion of hepatocyte and thus have less functional hepatic capacity, the release of miRNAs upon damage might be lower than in patients with higher amount of healthy liver tissues (26). Furthermore, the explain for increased miR-21-5p level in acute HCV infection is increased hepatic regeneration, as an in vivo study by Chen et al. who demonstrated that miR-21 is a critical regulator of liver regeneration and its upregulation contributed to hepatocyte proliferation by targeting PTEN (27). In the current study, the serum level of miR-196-5p was significantly upregulated in (AHC), (HCV-LC) and (SVR) patients compared to healthy control. On the other hand, no significant difference was shown in the expression of miRNA-196-5p between either (CHC vs. control) or (AHC vs. CHC, HCV-LC and SVR) groups respectively. These results were compatible with a previous study that found the serum levels of miRNA-196a were significantly decreased in chronic hepatitis C (CHC), they explained it by stating that the HCV core protein reduces the expression of miR-196a (28,29). Also, they were observed that serum miRNA-196 was found to be significantly higher in the treated group than in the control group. This was explained by the fact that miR-196, which is induced by INF-β, targets the HCV NS5A protein, reducing its replication. HCV inhibits the IFN pathway, which in turn causes downregulation of miR-196 expression. However, the results of this study disagreed with a study by Hussain et al., (30) who reported downregulation of miR-196 expression in recently infected HCV than control group but it was compatible with our results in that miR-196 was significantly elevated in treated patients and decreased in CHC patients. The explanation for this difference in miR-196a expression levels was that in recently diagnosed HCV-infected patients, HCV core protein decreased the expression of miRNA-196a while significantly increased in the treated patients (30). The increased serum miR-196-5p in the acute and SVR groups at similar levels gives an indication for the presence of a strong interferon immune response against the virus, while its decreased level in CHC and HCV-LC groups compared to AHC and SVR groups indicates weakened interferon immunity, with the lowest level in healthy control indicating the absence of underlying disease that induces interferon immune response. The results of Spearman's rank correlation analysis showed that there was significant negative and moderate correlation between serum miR-196-5p and age in SVR group (r=-0.722, P= 0.018), this result indicate that younger aged patients had better interferon response that increases the expression of miR-196-5p gene and this response was obvious in SVR group which showed better response to antiviral treatments at younger ages. The significant negative moderate correlation between miRNA-196-5p with serum TG and VLDL shown in our study in acute HCV group and significant negative moderate correlation with serum TG in HCV induced LC group can be explained by upregulated miRNA-196-5p by high levels of HCV and hepatic inflammation in these groups directly targets the transcription factor involved in lipid metabolism, fork head box O1 (FOXO1), leading to its down-regulation resulting in lipid accumulation and HCV induced

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cell proliferation (6). This study showed no significant correlation between serum miRNA-21-5p and serum liver function tests in contrary with previous studies (25, 31) who revealed positive correlation of miR-21 with ALT and AST serum levels in all patients and in both CHC and HCC groups, also disagreed with a study by (32) who reported that there was a significant positive correlation between miRNA-21 and ALT in chronic liver disease group. These discrepancies between results may be related to the underlying disease that HCV patients suffer from in our study, affecting the mechanism of microRNA-21-5p action in these patients. The diagnostic potential of microRNAs in different HCV groups was determined by ROC analysis, it revealed that miRNA-21-5p was excellent for diagnosing acute and chronic hepatitis C with (AUC= 1.000, sensitivity=100% and specificity=100% for both groups). The results in the current study were consistent with previous study that showed serum miRNA-21-5p could be a useful biomarker to differentiate chronic hepatitis from healthy individuals (29). However, miR-21 is not specific for HCV infection and it is elevated in cancers with high sensitivity (33). The ROCs analyses in the current study also indicated that serum miRNA-196-5p was excellent marker for the diagnosis and differentiation patients' groups from healthy group with higher accuracy in HCV-LC group (AURC= 0.886, sensitivity=80% and specificity=100 %) followed by SVR group (AURC= 0.871, sensitivity=70% and specificity=100%) and AHC group (AURC= 0.870, sensitivity=73% and specificity=100%) with lesser accuracy in CHC group (AURC= 0.725, sensitivity=54% and specificity=100%). These results were compatible with the study conducted earlier revealing an AUC for serum miRNA-196 of 0.8278 with 82.05% sensitivity and 76.19% specificity in discriminating healthy controls from HCV-infected samples, also found a significant increase in the serum level of miRNA-196a after treatment with antiviral drugs (30).

Conclusion

Increased serum miR-21-5p and miR-196-5p expression in HCV patients implicated in lipid dysregulation within hepatocytes and monitoring this correlation might be used as biomarker of disease progression and liver dysfunction.

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Ethical clearance: The Iraqi Ministry of Health officially approved this study at April/ 2024 and was provided by the Ethical approval Guideline number 3/1970 from the Medical Ethics Committee on human research of the Middle Technical University, College of Health and Medical Technology (Baghdad, Iraq).

Conflict of interest: None.

Use of Artificial Intelligence (AI): The authors state they did not use any generative AI tools for creating or editing the manuscript's language.

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ارتباط الرنا الدقيق بوظائف الكبد ومستويات الدهون لدى مرضى التهاب الكبد الوبائي سي كمؤشرات تشخيصية

[١] لبنى حسين مرشد، [١] جليل نجاح جليل، [١] عذراء زيدان حسن، [٢] صفاء عبد الكريم عبد الرزاق

الملخص

الخلفية: تتدخل الرنا الدقيق في تعديل مراحل مختلفة من دورة حياة فيروس إلتهاب الكبد الوبائي نوع سي وتم التعرف على أنواع معينة من الرنا الدقيق التي تختل تنظيمها أثناء الإصابة بفيروس إلتهاب الكبد الوبائي ويخدم كمتغير أساسي أثناء العلاج المضاد للفيروس.

الأهداف: لفهم دور الرنا الدقيق في المصل (miR-21-5p, miR-196-5p) وارتباطها مع وظائف الكبد والدهون كعلامات تشخيصية لدى المرضى الذين يعانون من مراحل مختلفة من فيروس إلتهاب الكبد سي.

المرضى والطرق: شارك في الدراسة ١٥٠ شخصًا - ١٠٠ مصاب بفيروس التهاب الكبد سي و ٥٠ شخصًا يتمتعون بصحة جيدة كمجموعة مراقبة. تم تقييم مستويات مصل المرضى والمجموعة الضابطة من miR-21-5p وmiR-196-5p باستخدام تفاعل البلمرة المستمر الكمي في الوقت الحقيقي (qRT-PCR)، وتم قياس الاختبارات الكيميائية الحيوية باستخدام محلل الكيمياء الحيوية التلقائي القياسي (Thermo Fisher 240 V Indiko Plus محلل الكيمياء السريرية، ولاية كيرالا، الهند).

النتائج: أظهرت الرنا الدقيق miR-21-5p ارتفاعا في مجموعتي التهاب الكبد الوبائي سي الحاد والمزمن, بينما أظهرت miR-196-5p ارتفاعا في مجموعات التهاب الكبد الوبائي سي الحاد وتليف الكبد الناتج عن فيروس التهاب الكبد سي ومجموعة الإستجابة المستدامة للعلاج مقارنة بالمجموعة الضابطة. الحد الأعلى لمستويات الدهون السيئة في المصل كانت في مجموعة الإستجابة المستدامة للعلاج بينما كانت الحد الأدنى في مجموعة تليف الكبد الناتج عن فيروس إلتهاب الكبد الوبائي نوع سي, وكانت مستويات فحوصات وظائف الكبد الأعلى في مجموعة فيروس إلتهاب الكبد الوبائي سي الحاد. يرتبط miR-196-5p سلبًا بالدهون الثلاثية والكوليسترول المنخفض الكثافة جدا في مجموعة فيروس التهاب الكبد الوبائي سي الحاد وتليف الكبد الناتج عن فيروس التهاب الكبد الوبائي سي. أظهر منحنى ROC أعلى حساسية ونوعية لـ miR-21-5p في مجموعات التهاب الكبد الفيروسي سي الحاد والمزمن، بينما كانت الحساسية والنوعية الأعلى لـ miR-196-5p في مجموعات التهاب الكبد الفيروسي سي الحاد وتليف الكبد الناتج عن فيروس التهاب الكبد الوبائي سي والإستجابة المستدامة للعلاج.

الاستنتاج: تشير زيادة التعبير عن الرنا الدقيق miR-21-5p وmiR-196-5p في مصل المرضى المصابين بفيروس التهاب الكبد الوبائي سي إلى تورطهم في خلل تنظيم الدهون داخل الخلايا الكبدية، وقد يتم استخدام مراقبة هذا الارتباط كعلامة حيوية لتطور المرض واختلال وظائف الكبد.

الكلمات المفتاحية: التهاب الكبد الوبائي، miR-21-5p، miR-196-5p، المصل، العلامات الحيوية.

المؤلف المراسل: لبنى حسين مرشد

الايميل: edc4005@mtu.edu.iq

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[١] كلية التقنيات الصحية و الطبية، الجامعة التقنية الوسطى، بغداد، العراق.

[٢] مستشفى الجهاز الهضمي التعليمي، دائرة صحة مدينة الطب، وزارة الصحة العراقية، بغداد، العراق.