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ASSOCIATION OF MIR-192 EXPRESSION WITH HEPATITIS C VIRUS INFECTION: A CASE-CONTROL STUDY

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ABSTRACT

Background and Objectives: Infection with the hepatitis C virus (HCV) is a major threat to global health that can cause cirrhosis, hepatocellular carcinoma, progressive fibrosis, chronic inflammation, and hepatic steatosis. MicroRNAs (miRNAs) are post-transcriptional regulators and mediators of viral pathogenesis. The purpose of this study is to investigate the relationship between liver function during HCV infection and the expression profile of miR-192. **Methods:** Seventy healthy controls and 70 HCV patients participated in a case-control study. Serum biochemical markers-including albumin, ALP, ALT, AST, and total serum bilirubin (TSB)-were evaluated using standard clinical assays. The $2^{-\Delta\Delta CT}$ method was used to measure the relative gene expression of miR-192 using total RNA extraction and quantitative real-time PCR (RT-qPCR). **Results:** There were no statistically significant ($P > 0.05$) age or gender distribution differences between the cohorts. Biochemical profiling revealed significant elevations in serum liver enzymes and TSB, as well as a significant decrease in serum albumin ($P < 0.05$) among patients. Molecular expression analysis showed that the patient group had a statistically significant downregulation of miR-192 in comparison to healthy controls (patient mean fold change: 0.285 versus control mean fold change: 1.00; $P < 0.05$). Gender-stratified analyses showed significant variations in fold change values between male and female subgroups. The correlations between clinical liver function parameters and the expression levels of miR-192 were analysed using Pearson correlation test. Conclusion: The considerable downregulation of miR-192 in HCV patients indicates its potential as a non-invasive biomarker to evaluate liver damage.

INTRODUCTION

Infection with hepatitis C virus (HCV) is a serious global public health problem with chronic hepatitis, hepatic steatosis, progressive fibrosis, cirrhosis and hepatocellular cancer as frequent sequelae [1]. The pathogenesis of HCV is believed to be associated with strong inflammatory responses and structural histological abnormalities of the liver tissue induced by complex molecular interactions between the virus and the host cells [2]. MicroRNAs (miRNAs) are small non-coding RNAs (19-22 nucleotides in length) that have emerged as major post-transcriptional regulators of gene expression [3] and key mediators of viral pathogenicity [4].

Liver-enriched miRNAs are good candidates for non-invasive diagnostic biomarkers because they are released into the circulation after cellular injury and are typically dysregulated in chronic liver disorders [4]. One such example is miR-192, a key hepatic-enriched microRNA which is strongly correlated with liver inflammation and fibrogenic pathways [5]. miR-192 expression is markedly changed in HCV infection and intimately interacts with profibrogenic signalling cascades such as TGF-β1 pathways [6]. It has also been demonstrated to modulate viral replication dynamics and host cellular responses [7]. Changes in miR-192

levels in patient serum and hepatic tissue correlate with the course of liver damage and disease severity [8]. The exact correlation of baseline miR-192 expression levels with HCV infection is yet to be known [9].

This study was designed to analyse the expression profile of miR-192 and to evaluate its association with hepatitis C virus infection so as to better understand the molecular function of miR-192 in viral pathogenesis [6,9].

MATERIALS AND METHODS

Study Design and HCV Molecular Detection

The aim of this case control study is to determine the connection of hepatitis C virus (HCV) infection and expression profile of miR-192. Venous blood samples (5 ml) were obtained from each participant in EDTA tubes. Screening and confirmation of HCV infection in blood samples to identify patients and healthy control groups was performed using specialised diagnostic techniques including enzyme-linked immunosorbent assay (ELISA) and RT-PCR confirmation for viral load and active infection.

Biochemical and Clinical Analysis

Patients were clinically and laboratory examined by specialist physicians to evaluate the length and severity of liver cirrhosis. Tests were performed to identify chronic diseases such as diabetes, hypertension and viral hepatitis that led to the development of liver fibrosis. Hepatic status and liver injury were assessed through evaluation of serum liver enzymes and biochemical markers including total serum bilirubin (TSB), alkaline phosphatase (ALP), alanine aminotransferase (ALT), aspartate aminotransferase (AST) and albumin. Sample analysis and quantification were performed according to the regular operation procedures of the FUJIFILM DRI-CHEM system.

Quantitative Real-Time PCR (RT-qPCR)

Total RNA was extracted from blood samples using the TRIzol® Reagent kit in accordance with the manufacturer's instructions. A Nanodrop Spectrophotometer was used to measure the absorbance at 260/280 nm to determine the concentration and purity of the extracted RNA. To eliminate trace amounts of genomic DNA, the extracted RNA was treated with DNase I enzyme using a DNase I enzyme kit (Promega, USA).

GoTaq® qPCR Master Mix was used in the quantitative real-time PCR procedure for gene expression analysis. Specific primers for miR-192 (MIMAT0000222) and the housekeeping gene GAPDH (NM_001256799.3) were designed using the Primer3 design tools, based on sequences from the Sanger Center's miRNA database registry and the NCBI database. In addition to a universal stem-loop RT primer, specific forward and reverse primers were used for miR-192 (Forward: 5′-CTGACCTATGAATTGACAGCC-3′; Reverse: 5′-GTCGTATCCAGTGCAGGG-3′) and GAPDH (Forward: 5′-TCACCAGGGCTGCTTTAC-3′; Reverse: 5′-TGACGGTGCCATGGAATTTG-3′). These primers were supplied by Macrogen Company (South Korea). The components of the qPCR master mix and template were loaded into qPCR strip plates, mixed using an Exispin vortex centrifuge for three minutes, and placed into a Miniopticon Real-Time PCR system. The thermal cycling conditions included a five-minute initial denaturation step at 95 °C, followed by 45 cycles of denaturation at 95 °C for 20 seconds, and annealing/extension/detection at 60 °C for 30 seconds.

Statistical Analysis

The ΔΔCT method (fold change = $2^{-\Delta\Delta CT}$), as described by Livak and Schmittgen, was used to calculate the relative quantification of gene expression levels in comparison to the endogenous control gene. Data analysis was carried out using Excel 2010 and the Statistical Package for the Social Sciences (SPSS), version 22. Differences with $P < 0.05$ were considered statistically significant.

RESULTS

The demographic characteristics of the study participants are presented in Table 1. There were 70 patients in the HCV group and 70 healthy individuals in the control group. Neither the age distribution nor the gender ratios showed statistically significant differences between the two groups ($P > 0.05$). The patients' mean age was 56.33 ± 8.89 years (range: 18–78 years), while the control group had a comparable mean age of 55.6 ± 10.19 years (range: 19–78 years; $P = 0.851$). The patient cohort comprised 36 males (51.4%) and 34 females (48.6%), while the control cohort comprised 39 males (55.7%) and 31 females (44.3%), with non-significant P values of 0.692 and 0.577, respectively.

Table 01: Demographic analysis of study participants.

Data	**Patients (n = 70)**	**Controls (n = 70)**	**P value**
Age range (years)	18–78	19–78	
Mean ± SD	56.33 ± 8.89	55.6 ± 10.19	0.851
Gender	**N (%)**	**N (%)**	**P value**
Males	36 (51.4%)	39 (55.7%)	0.692
Females	34 (48.6%)	31 (44.3%)	0.577
P value (within group)	0.799	0.058	
Total	**70**	**70**	

SD, standard deviation.

All of the tested biochemical markers showed statistically significant differences between the patient and control groups (Table 2). Serum albumin levels were significantly lower in patients (mean: 1.13 ± 0.21 g/dL) compared to controls (5.93 ± 0.53 g/dL). In contrast, the patient group showed significant elevations in hepatic enzymes and bilirubin. ALP levels were elevated to a mean of 380.3 ± 85.9 U/L in patients versus 102.84 ± 13.2 U/L in controls. Similarly,

ALT (52.2 ± 9.88 U/L vs. 17.09 ± 4.37 U/L) and AST (33.77 ± 9.93 U/L vs. 9.52 ± 2.54 U/L) were significantly higher in patients. TSB levels were also markedly elevated in patients (22.5 ± 7.51 mg/dL) compared to controls (2.10 ± 0.09 mg/dL).

Table 02: Comparison of liver function test parameters between HCV patients and healthy controls.

Tests	Patients (Mean ± SD)	Controls (Mean ± SD)	95% CI	P value
Albumin (g/dL)	1.13 ± 0.21	5.93 ± 0.53	−4.94 to −4.66	< 0.001*
ALP (U/L)	380.3 ± 85.9	102.84 ± 13.2	257.10 to 297.82	< 0.001*
ALT (U/L)	52.2 ± 9.88	17.09 ± 4.37	32.58 to 37.64	< 0.001*
AST (U/L)	33.77 ± 9.93	9.52 ± 2.54	21.85 to 26.69	< 0.001*
TSB (mg/dL)	22.5 ± 7.51	2.10 ± 0.09	18.64 to 22.16	< 0.001*

*Statistically significant (P < 0.05). SD, standard deviation; CI, confidence interval.

Molecular study of the gene expression of miR-192 is shown in Table 3 and Figure 2 with significant variations between groups. The mean cycle threshold (CT) of miR-192 in patients was 25.96 with GAPDH mean CT of 22.27, hence the mean ΔCT was 3.69. The mean miR-192 CT of the control group was 28.76 and the mean GAPDH CT was 26.88, with a meanΔCT of 1.88. The average ΔΔCT for the sick group was 1.81 and for the control (calibrator) group was 0.00. Relative quantification revealed a mean fold change (2−ΔΔCT) of 0.285 in the sick group showing a substantial down-regulation in comparison to a fold change of 1.00 in the healthy control group (calibrator) (P < 0.05).

Table 03: Comparison of miR-192 gene expression between HCV patients and healthy controls.

Group	Mean CT (miR-192)	Mean CT (GAPDH)	Mean ΔCT	Mean ΔΔCT	Fold Change ($2^{-\Delta\Delta CT}$)
Patients	25.96	22.27	3.69	1.81	0.285
Controls	28.76	26.88	1.88	0.00	1.00*

*Statistically significant difference compared with patients (P < 0.05).

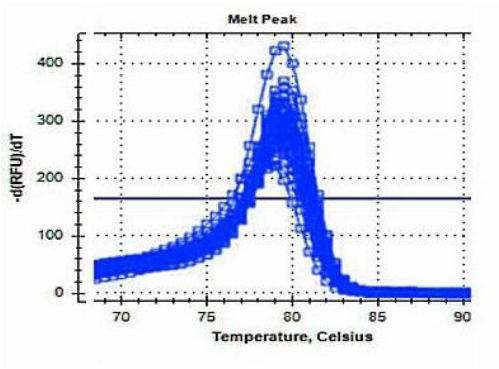

Figure 01: Real-time PCR melting curve analysis of miR-192 gene expression showing qPCR specificity at 80.07 °C.

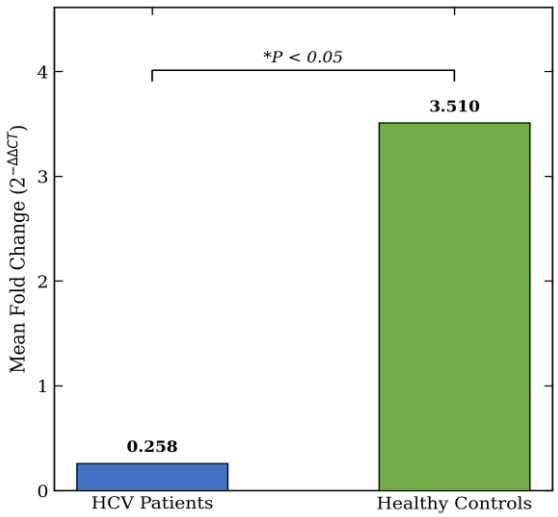

Figure 02: Comparison of miR-192 fold change ($2^{-\Delta\Delta CT}$) between HCV patients and healthy controls. *P < 0.05.

The gender-stratified analysis of miR-192 expression (Table 4 and Figure 3) revealed significant differences between male and female participants in both groups. Among male subjects, the mean fold change was 0.658 ± 0.14 in patients and 2.73 ± 0.16 in controls (P = 0.042). Among female subjects, the patient group showed a mean fold change of 0.489 ± 0.03 versus 3.801 ± 0.21 in the control group (P = 0.036). The difference between gender-specific fold change values within the patient cohort was also statistically significant (P = 0.047), while the difference within the control cohort did not reach significance (P = 0.061).

Table 04: Gender-stratified analysis of miR-192 gene expression in HCV patients and controls.

Gender	Patients FC ($2^{-\Delta\Delta CT}$) ± SD	Controls FC ($2^{-\Delta\Delta CT}$) ± SD	P value
Males	0.658 ± 0.14	2.73 ± 0.16	0.042*
Females	0.489 ± 0.03	3.801 ± 0.21	0.036*
P value	0.047*	0.061	

*Statistically significant (P < 0.05). FC, fold change; SD, standard deviation.

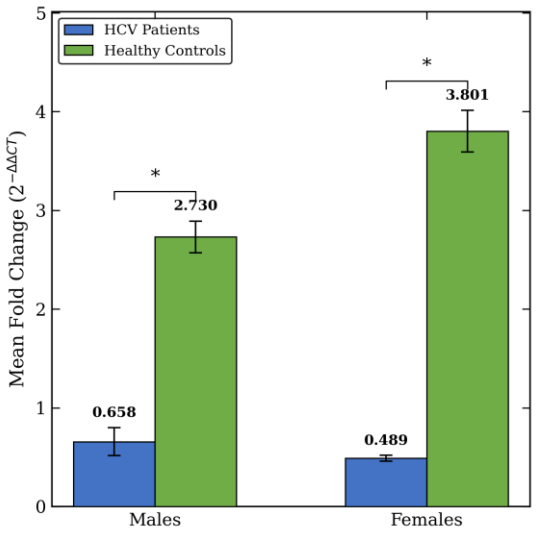

Figure 03: Gender-stratified comparison of miR-192 fold change ($2^{-\Delta\Delta CT}$) between HCV patients and healthy controls. Error bars represent standard deviation. *P < 0.05.

The Pearson correlation analysis of miR-192 expression with liver function tests parameters in HCV patients was shown in Table 5 and Figure 4. The found association coefficients were albumin (r=-0.013), ALP (r=0.151), ALT (r=0.019), AST (r=0.042) and TSB (r=0.090). None of these associations were statistically significant (all P > 0.05) demonstrating that the fold change of miR-192 did not correlate linearly with any single liver function parameter in the patient group.

Table 05: Pearson correlation (r) between miR-192 expression and liver function tests in HCV patients.

Liver Function Test	Pearson Correlation (r)	P value
Albumin (g/dL)	−0.013	**0.915**
ALP (U/L)	0.151	**0.212**
ALT (U/L)	0.019	**0.876**
AST (U/L)	0.042	**0.730**
TSB (mg/dL)	0.090	**0.459**

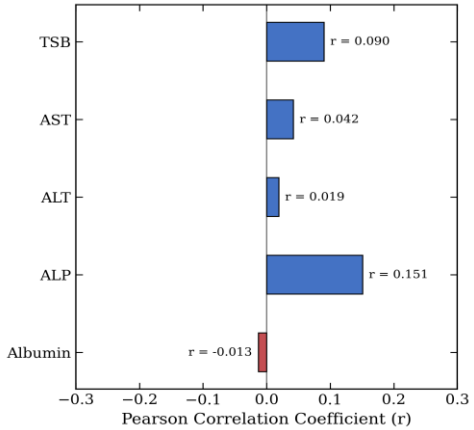

Figure 04: Pearson correlation coefficients between miR-192 fold change and liver function test parameters in HCV patients.

DISCUSSION

The demographic data reported in Table 1 shows the patient and control groups have a comparable age and gender distribution (P > 0.05). This absence of statistically significant demographic differences is considered an important methodological strength since it rules out any confounding bias linked to age and sex. Therefore, the subsequent biochemical and molecular aberrations seen are probably caused by pathogenic mechanisms rather than by variations between the baseline population [10,11]. These structural alignments are in line with typical epidemiological parameters in similar clinical cohorts studying the progression of chronic liver disease; matched demographic baselines ensure high internal validity and reproducibility across multi-center studies [12,13].

The classical biochemical hallmark of severe hepatocyte membrane damage and cytolysis and decreased hepatic synthetic ability is reflected at the clinical biochemistry level (Table 2) by marked elevations of serum liver enzymes, such as ALP, ALT and AST, and TSB together with a significant decrease in serum albumin (P < 0.05). The clinical results are very similar to theseinsights emphasized by Giannini et al. (2005), who noted that systemic enzyme alterations reliably track the progression of hepatocellular damage [14].

At the molecular standard, the expression profile of miR-192 (Table 3) shows important regulatory abnormalities. MicroRNAs are important post-transcriptional regulators of liver homeostasis, inflammation and fibrogenesis, but the dysregulation observed suggests a breakdown of normal defensive networks in disease aetiology [15]. The importance of extracellular and circulating microRNAs in monitoring progression of liver disease is further supported by Roderburg and Luedde (2014) and recent clinical studies by Abdelaziz et al. (2026) [11,16]. Moreover, these molecular patterns are consistent with findings from larger cohorts investigating chronic viral hepatitis sequelae as evaluated by Ullah et al. (2025), and particular biomarker studies emphasising the significance of non-coding RNAs in monitoring fibrosis stage [17].

Moreover, gender-stratified analysis (Table 4) revealed substantial differences in fold change values within and across groups (P < 0.05), suggesting a pattern of sexual dimorphism. It implies that sex-specific hormonal and genetic modulators are actively involved in the regulation of microRNAs expression and the rate of hepatic damage, in agreement with literature on gender-dependent differences in viral hepatitis pathways [18,19]. However, the Pearson correlation analysis (Table 5) did not find any statistically significant linear link between the expression of miR-192 and each of the indicators of liver function (all P > 0.05). This indicates that, whereas miR-192 is variably expressed at the group level, its regulation may be mediated through complicated, non-linear processes rather than direct proportionate coupling with

particular biochemical indicators. Li et al. (2022) shown that liver-enriched microRNAs regulate intracellular signalling pathways such as Akt/mTOR cascades [20]. The absence of simple linear correlations may reflect the multi-target nature of miRNA-mediated regulation.

Several previous studies suggest that miR-192 may act as an antiviral agent in the host by inhibiting hepatitis C virus replication and may have a promising therapeutic role in treating HCV infection. Furthermore, the overexpression of miR-192 and miR-29a in HCV-infected patients may be attributed to their role in regulating fibrosis and inflammation, two hallmarks of chronic liver disease [21,22]. miR-192 and miR-29a may serve as potential non-invasive biomarkers for diagnosing fibrosis progression following HCV-induced chronic liver disease, as supported by previous studies. The reasons for the discrepancies in miR-192 and miR-29a levels between liver tissue and serum samples remain unknown. Researchers have hypothesized that differences in sample preparation and analytical procedures may contribute to these discrepancies. Therefore, more research are needed to elucidate the reasons behind these differences and to establish standardised methods for measuring miRNA concentrations in different sample types [23,24].

Our study provides further data to support the possible application of miR-192 as a biomarker of liver disease. The present biomarkers may be useful for diagnosing the progression of fibrosis in patients with chronic liver disease with hepatitis C infection. However, further studies are required to evaluate the potential of these biomarkers for diagnostic and therapeutic purposes and to standardise the methodologies of miRNA levels detection from diverse types of samples. Recent studies have assessed the diagnostic value of miR-192 as a biomarker in hepatitis C patients, although the results remain limited [24–27].

LIMITATIONS

Several limitations of this study need to be acknowledged. First, the sample size is rather small (n = 70 each group), which may restrict the statistical power in detecting weak relationships. Second, using GAPDH as an endogenous control for stem-loop RT-qPCR of mature miRNAs is a methodological issue, since GAPDH is an mRNA-class transcript and may not adequately represent the small-RNA fraction in serum samples. A validated small-RNA normaliser would have been preferable. Third, the study was cross-sectional, and so was not able toanalyse temporal changes in miR-192 expression as the disease progresses. Fourth, HCV genotype information was not disclosed and may impact the miRNA expression profiles. Finally, the study did not have individuals with other chronic liver disorders for comparison, which would have been helpful to confirm specificity of miR-192 as an HCV-related biomarker. Future studies should confirm these findings in bigger cohorts, longitudinal investigations and using verified small-RNA normalisers.

CONCLUSION

The results of this investigation showed a significant reduction in the expression level of miR-192 in chronic HCV patients as compared to the healthy subjects. This downregulation of miR-192 could be due to disrupted post-transcriptional regulatory networks during illness progression. The observed molecular alterations correlate with classical biochemical markers of hepatocyte injury, cytolysis and reduced synthetic liver function. The gender-stratified results also show significant sex differences in the amount of miR-192 expression, suggesting the existence of potential hormonal or genetic variables regulating the biological response. In conclusion, our findings warrant future study of miR-192 as a potential non-invasive marker for assessment of hepatic injury in HCV patients.

ETHICS STATEMENT

The current study was approved by the Research Ethics Committee at the University of Al-Qadisiyah (Approval No. QU-2122-5-7-2025). All techniques performed in this research were in compliance with the ethical norms of the institutional research committee and with the 1964 Declaration of Helsinki and its later amendments. All participants gave their written informed consent before taking part in the study.

AUTHOR CONTRIBUTIONS

Lamyaa Sabeeh Abdullah: Conceptualisation, Methodology, Investigation, Writing – Original Draft, Formal Analysis. Supervision, Writing-Review & Editing, Validation [Author 2]. [Author 3]: Resources, Data Curation. All writers read and approved the final manuscript.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are accessible from the corresponding author upon reasonable request.

DECLARATION OF INTEREST

Conflict of interest we declare we have no conflict of interest.

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