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Reduced PTEN mRNA Expression in Blood of Patients with Chronic Hepatitis C Virus Infection: A Case-Control Study

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ABSTRACT

Background & Objective: Chronic hepatitis C virus (HCV) infection remains a major global health concern and is a leading cause of liver cirrhosis and hepatocellular carcinoma (HCC). The tumor suppressor gene phosphatase and tensin homolog (PTEN) play a crucial role in regulating the PI3K/AKT pathway, a key signaling cascade involved in cell survival and proliferation. This study aimed to investigate the expression of PTEN mRNA in the whole blood of patients with chronic HCV infection compared to healthy individuals.

Materials & Methods: In this case-control study, blood samples were collected from 50 patients with chronic HCV infection and 50 healthy controls. Total RNA was extracted from whole blood and converted to cDNA. PTEN expression was quantified using SYBR Green-based Real-time PCR, with GAPDH as the internal control. Data were analyzed using the 2^-ΔΔCt method and appropriate statistical tests.

Results: PTEN expression was significantly downregulated in the patient group compared to controls (fold change = 0.085; $P< 0.0001$), indicating an approximately 8.5-fold reduction. Age-stratified analysis revealed the lowest PTEN expression in patients over 50 years of age. These findings suggest systemic molecular alterations in HCV-infected individuals and a potential age-related pattern of PTEN suppression.

Conclusion: The marked reduction of PTEN mRNA expression in chronic HCV patients highlights its potential role in HCV pathogenesis and progression to HCC. PTEN may serve as a promising biomarker and therapeutic target in chronic HCV infection.

Keywords: Hepatitis C virus, PTEN, Gene expression, Tumor suppressor, Real-time PCR, Hepatocellular carcinoma

1. Introduction

Hepatitis C virus infection affects more than 185 million people globally, posing a major public health burden (1). Chronic infection develops in approximately 75%–85% of those with acute HCV, and among these, 10%–20% gradually progress to liver cirrhosis. Of those with cirrhosis, 1%–5% develop hepatocellular carcinoma each year, the most common form of primary liver cancer and the third leading cause of cancer-related death worldwide (2, 3). Although the advent of highly effective direct-acting antivirals (DAAs) has transformed HCV treatment, the incidence, morbidity, and mortality associated with HCV-related HCC remain concerning. Notably, in 2021, over 150,000 deaths were attributed to HCV-associated HCC, the highest annual toll recorded to date (4).

Unlike hepatitis B virus (HBV), which integrates into the host genome, HCV-induced carcinogenesis is thought to be driven primarily by chronic inflammation, oxidative stress, and dysregulation of host signaling pathways. One of the key pathways involved in cell proliferation, survival, and apoptosis is the phosphatidylinositol 3-kinase/protein kinase B (PI3K/AKT) signaling cascade (5). This pathway is negatively regulated by the tumor suppressor gene PTEN, which dephosphorylates

phosphatidylinositol 3, 4, 5-trisphosphate (PIP3) to phosphatidylinositol 4,5-bisphosphate (PIP 2) (6). Indeed, PTEN inhibits signaling through the PI3K-Akt axis, thereby controlling growth factor signaling and acting as a potent tumor suppressor (7). Concerning hepatic metabolic disorders and cancer, there is growing evidence that underscores the significant role of PTEN in the progression of these diseases (8).

Given the pivotal role of PTEN in maintaining cellular homeostasis and preventing malignant transformation, investigating its expression in HCV-infected individuals may provide valuable insights into the molecular mechanisms underlying HCV-associated liver pathology (9). Although previous studies have investigated PTEN expression in hepatic tissues (10) or under in vitro conditions (11), data on PTEN mRNA expression in whole blood of chronic HCV patients are limited. Given the accessibility and minimally invasive nature of whole blood collection, such an approach may offer valuable insights into the systemic molecular alterations associated with HCV pathogenesis. This study aimed to evaluate the expression levels of PTEN in patients with chronic HCV infection compared to healthy individuals.

2. Materials and Methods

2.1 Study Population

This case-control study evaluated PTEN expression in patients with chronic HCV infection compared with healthy individuals. Blood samples were collected from both the case and control groups at the Keyvan Virology Laboratory in Tehran, Iran, between February 2023 and August 2024, according to predefined inclusion and exclusion criteria. The case group included adult patients with chronic HCV infection, defined as the presence of HCV RNA in serum for more than six months. Chronicity was confirmed by an infectious disease specialist based on laboratory records and clinical evaluation. Exclusion criteria for the case group included a history or clinical diagnosis of hepatocellular carcinoma (HCC), co-infection with HBV or HIV, presence of other chronic liver diseases, malignancies, or use of immunosuppressive medications. The control group comprised healthy individuals who tested negative for anti-HCV antibodies and had normal levels of liver enzymes (ALT, AST).

2.2 Sample Collection and RNA Extraction

Peripheral blood samples were collected into EDTA-containing tubes and transported to the laboratory under appropriate conditions. Total RNA was extracted from whole blood using an RNA extraction kit (Yekta Tajhiz, Iran), following the manufacturer's instructions. The quality and quantity of the extracted RNA were assessed using a Nanodrop spectrophotometer (Nabi, South Korea).

2.3 cDNA Synthesis and Real-Time PCR

cDNA synthesis was performed using a cDNA synthesis kit (Pars Tous, Iran) according to the manufacturer's protocol. The expression of the reference gene GAPDH and the target gene PTEN was evaluated using SYBR Green-based Real-Time PCR. Primer specificity was confirmed by melt curve analysis.

Real-time PCR reactions were performed using SYBR Green Master Mix (Amplicon, Denmark) in a final volume of 20 µL, containing 10 µL of SYBR Green master mix, 1 µL of each primer (10 µM), 4 µL of cDNA, and 4 µL of nuclease-free water. The thermocycling conditions included an initial denaturation at 95°C for 10 minutes, followed by 45 cycles of denaturation at 95°C for 15 seconds, annealing at 55°C for 30 seconds, and extension at 72°C for 30 seconds. All reactions were run in triplicate. The relative expression level of the PTEN gene was calculated using the 2^-ΔΔCt method, with GAPDH as the internal control.

Table 1. Primer sequences

Gene	Sequence (5' → 3')	Length	Annealing temperature (°C)	Reference
F: PTEN	ACCAGTGGCACTGTTGTTTC	20	55°C	(12)
R: PTEN	TTAGCTGGCAGACCACAAAC	20		
F: GAPDH	TCAGCCGCATCTTCTTTTGC	20	55°C	
R: GAPDH	TTAAAAGCAGCCCTGGTGAC	20		

2.4 Statistical Analysis

Statistical analysis was performed using SPSS software version 26. Descriptive statistics, including means, standard deviations, and frequencies, were used to summarize the data.

The normality of data distribution was assessed using the Shapiro-Wilk test. For comparisons of gene expression levels between groups, independent samples t-tests were applied when normality
assumptions were met; otherwise, the non-parametric Mann-Whitney U test was used.

A *P*-value of less than 0.05 was considered statistically significant.

3. Result

A total of 100 participants were enrolled in this study, including 50 patients with chronic HCV infection and 50 healthy individuals as the control group.

The mean age of participants was 48.10 ± 12.17 years in the patient group and 30.38 ± 19.88 years in the control group.

Among HCV-infected individuals, 64% (32/50) were male, and 36% (18/50) were female. In contrast, the control group consisted of 50% males and 50% females.

3.1 Gene Expression Analysis

The Mean Ct value of PTEN expression was 25.03 in the control group and 27.82 in the patient group. The mean Ct value for GAPDH was 24.35 in the control group and 23.59 in the patient group. The calculated ΔCt and ΔΔCt values are provided in Table 2. Using the 2^-ΔΔCt method, the relative expression of PTEN in the patient group was found to be approximately 0.085, which corresponds to an 8.5-fold decrease in expression compared to the control group. This indicates a statistically significant downregulation of PTEN in chronic HCV patients ($p < 0.0001$) (Table 2). According to the 2^-ΔΔCt approach, a value less than 1 reflects gene downregulation, whereas values greater than 1 indicate upregulation. Thus, our data suggest suppressed PTEN expression in the case group (Figure 1).

Table 2. Comparative analysis of PTEN gene expression between HCV patients and healthy controls

Value		Up/down	*P*-value (t test)
PTEN control (Gene expression ratio)	1.03		
PTEN case (Gene expression ratio)	1.17	Down	*P*<0.0001
PTEN control (ΔCT)		0.674	
PTEN case (ΔCT)		4.226	
Mean ΔΔCt		3.548	
2^-ΔΔCt		0.085	

Note: ΔΔCT = (ΔCT_case - ΔCT_control)

ΔCT Control Group (PTENcontrol - GAPDHcontrol)

ΔCT Case Group (PTENcase - GAPDHcase)

$2^{-\Delta\Delta Ct} = 2^{-}$ ((Ct target, case−Ct reference, case) − (Ct target, control−Ct reference, control))

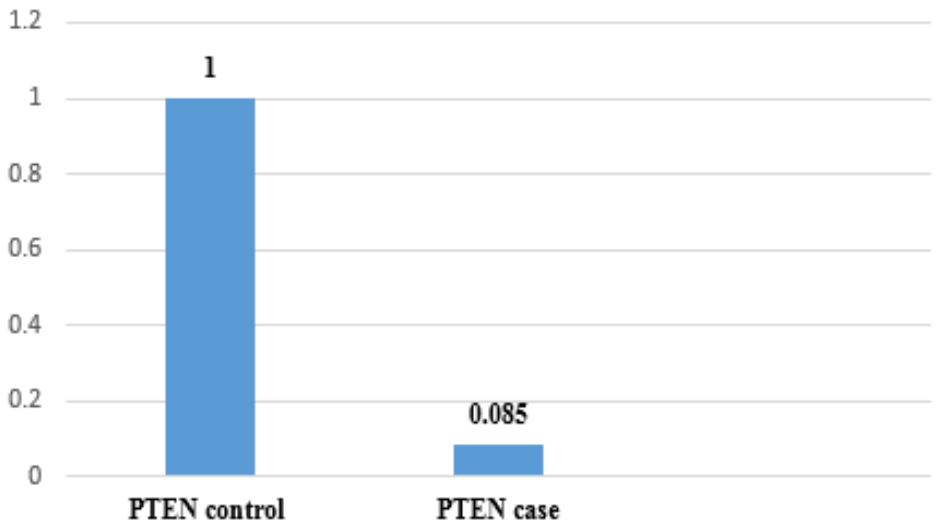

Figure 1. PTEN Gene Expression in chronic HCV infection and healthy controls. Gene expression was measured by qRT-PCR and normalized using ΔΔCt method. The 2^-ΔΔCt values indicate that PTEN expression is significantly downregulated in chronic HCV patients (0.085) compared to controls (1.0), $P < 0.0001$. **(Prepared by Authors, 2026)**.

Comparison of PTEN expression among different age groups in the patient population, as shown in Table 3, revealed variation in gene expression. The highest expression ratio was observed in the 30–50-year age group (1.155), followed by the <30-year group (1.119), and the lowest expression was noted in the >50-year group (1.061). Although the differences in PTEN expression across age groups in our study were not statistically significant (P=0.23) but these results suggest a potential age-related pattern of PTEN expression among HCV-infected individuals.

Table 3. Comparison of PTEN ratios between different age groups.

Age Group	PTEN ratio
30-50	1.155
<30	1.119
>50	1.061

4. Discussion

Viruses are the etiologic agents for more than 16-18% of tumors in humans (2). The chronic HCV infection has been strongly associated with liver inflammation, fibrosis, steatosis, and eventually the development of HCC (12). Despite the approval of anti-viral drugs, HCV infection continues to be a significant public health problem with HCC as the deadliest clinical outcome. The remaining risk for patients to develop HCC, even after sustained virologic response (SVR), appears to result from the persistent impact of HCV infection on liver homeostasis (13). The direct interaction of HCV RNA or viral proteins with oncogenic pathways in infected hepatocytes may still contribute to hepatocarcinogenesis (4).

Loss or downregulation of PTEN is frequently observed in a range of human cancers, including HCC, and is recognized for its role in tumor initiation, progression, and resistance to apoptosis. PTEN is a crucial tumor suppressor gene that regulates various cellular processes, including cell proliferation, apoptosis, and metabolism, primarily through its inhibitory effect on the PI3K/Akt signaling pathway (14). The PI3K/Akt signaling pathway plays a significant role in HCV infection and pathogenesis. HCV hijacks this pathway to promote its entry, replication, and survival in infected cells. In contrast, PTEN, a negative regulator of the pathway, is also targeted by HCV to enhance these processes (15).

In the current study, we evaluated PTEN expression levels in whole blood samples from patients with chronic HCV infection compared to healthy controls. The results demonstrated a significantly lower expression of PTEN in the HCV-infected group, with a relative fold change of approximately 0.085 (i.e., an 8.5-fold reduction) as determined by the 2^-ΔΔCt method (P< 0.0001). This reduction in PTEN expression supports the notion that HCV infection disrupts tumor suppressor signaling and fosters a cellular environment conducive to malignant transformation. Our findings are in agreement with those of Cheng et al., who reported that HCV suppresses PTEN gene transcription and protein expression in hepatocytes, primarily through the viral NS5A protein (16). Additionally, Wu et al. demonstrated that the HCV core protein decreases PTEN levels by either inhibiting PTEN mRNA translation or activating the NF-κB pathway. The replication of wild-type HCV was associated with decreased PTEN protein levels (17). Recently, a study has shown that PTEN gene expression is significantly downregulated in patients with hepatitis C virus-induced hepatocellular carcinoma compared with healthy individuals. The authors reported that reduced PTEN expression was associated with more advanced liver disease, indicating its potential role not only in tumor initiation but also in disease progression (18).

Aging itself is associated with physiological changes, including reduced immune surveillance, chronic inflammation and epigenetic modifications (19, 20). In this regard, PTEN has been shown to play a crucial role in age-related diseases. Tait et al. reported that age-related changes in PTEN expression and activity can disrupt key cellular pathways, particularly the PI3K/AKT signaling pathway, leading to increased cellular senescence, impaired apoptosis, and higher susceptibility to malignancies. These findings suggest that the observed association between reduced PTEN expression and more advanced liver disease may, at least in part, result from age-related molecular changes that make hepatocytes more vulnerable and promote tumor progression in chronic HCV infection (21). Although the differences in PTEN expression across age groups in our study were not statistically significant, observed association between reduced PTEN expression and age also suggests that older patients may be at increased risk for more profound molecular alterations. However, further stratified studies are needed to explore the interaction between age, PTEN expression, and risk of HCV-related hepatocellular carcinoma.

Several studies have revealed the multifaceted role of PTEN in controlling various stages of the HCV life cycle. PTEN has been shown to inhibit HCV entry, translation, replication, and secretion, thereby acting as a broad-spectrum antiviral regulator against HCV infection (17). Zhang et al. reported that the HCV core protein downregulates PTEN expression at both mRNA and protein levels, while simultaneously activating downstream targets such as Akt and NF-κB, key players in cell survival and inflammation (22). Moreover, suppression of PTEN activity by HCV has been linked to altered cholesterol metabolism, resulting in the formation of large lipid droplets that facilitate increased virion assembly and release (11). The viral protein NS5A further contributes to PTEN suppression by activating the NF-κB and PI3K/Akt signaling pathways and disrupting apoptotic mechanisms. Notably, NF-κB can directly bind to the PTEN promoter to repress its transcription, suggesting a mechanistic link between NS5A activation and PTEN downregulation (16, 23).

In addition to protein-mediated mechanisms, microRNAs particularly miR-21-5p have also been implicated in the suppression of PTEN. HCV infection has been shown to upregulate miR-21-5p, which binds to PTEN mRNA and inhibits its expression.

Given its role in other metabolic liver conditions such as alcoholic and obesity-related fatty liver disease, miR-21-5p may represent a shared regulatory pathway contributing to PTEN loss in a range of chronic hepatic disorders (24). Wu et al. demonstrated that intracellular overexpression of PTEN-Long inhibits HCV replication and treatment with extracellular PTEN-Long protein inhibits HCV replication in a dose-dependent manner. Also, targeting the regulatory pathways of PTEN, such as NS5A signaling and miRNA modulation, may offer novel strategies for preventing disease progression in chronic HCV patients. Future studies are recommended to confirm these findings at the protein level and explore the longitudinal impact of PTEN suppression on clinical outcomes in HCV-infected populations (25). Interestingly, high PTEN expression was evaluated as a positive independent prognostic factor for the survival of HCV-positive cirrhotic HCC patients (8). One of the key limitations of our study is the exclusive assessment of PTEN at the mRNA level; protein expression and activity were not evaluated. Additionally, a relatively small sample size and lack of longitudinal data limit the generalizability of our findings. Future research should aim to validate these results through protein-level analyses, investigate the impact of DAAs on PTEN expression, and explore the role of miRNAs such as miR-21-5p in modulating PTEN in the HCV-infected liver microenvironment. Collectively, these findings emphasize the potential of PTEN as a diagnostic biomarker and therapeutic target in the management of chronic HCV infection and its complications.

5. Conclusion

This study provides evidence that chronic HCV infection significantly reduces PTEN expression, which may represent an important step in viral pathogenesis and oncogenesis.

The observed findings underscore the need for further investigation into PTEN as a diagnostic and therapeutic target in HCV-associated liver disease.

6. Declarations

6.1 Acknowledgments

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6.2 Ethical Considerations

The study protocol was approved by the Ethics Committee of Hamadan University of Medical Sciences (Ethics code: IR.UMSHA.REC.1401.278).

6.3 Authors' Contributions

Conceptualization, S.Sh; Sample collection, F.T.A and S.Sh and Sh.M; methodology, S.Sh and Sh.M; Analysis, A.Sh; writing original draft preparation, S.Sh and Sh.M; review and editing, F.A.J and R.A; supervision, Sh.M. All authors have read and agreed to the published version of the manuscript.

6.4 Conflict of Interest

The authors have no conflict of interests related to this publication.

6.5 Fund or Financial Support

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6.6 Using Artificial Intelligence Tools (AI Tools)

The authors were not utilized AI Tools.

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