Assessment of Thyroid Profile in Patients with Hepatitis C Virus Infection

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ABSTRACT

Background: Hepatitis C virus (HCV) infection remains a major public health concern in Pakistan, with potential impacts on thyroid function. Long-term HCV infection has been associated with thyroid dysfunction, but the extent of this relationship is not fully understood.

Objective: This study aimed to evaluate the thyroid hormone profile in HCV-positive patients to assess the prevalence of thyroid dysfunction in this population.

Methods: A cross-sectional study was conducted on 120 HCV-positive patients aged 18-70 years. Patients with liver cirrhosis, known thyroid disorders, or medications affecting thyroid function were excluded. Blood samples were collected, and thyroid hormones (TSH, FT3, FT4) were measured using an automated immunoassay system. Data were analyzed using SPSS version 25.0, with a p-value <0.05 considered statistically significant.

Results: The study found that 17.50% of patients had elevated TSH levels, while 11.66% and 10.83% had elevated FT3 and FT4 levels, respectively. The mean values for TSH, FT3, and FT4 were 1.15 ± 0.515 μIU/mL, 1.10 ± 0.351 pg/mL, and 1.12 ± 0.361 ng/dL.

Conclusion: The study demonstrated a significant association between HCV infection and thyroid dysfunction, highlighting the need for regular thyroid function monitoring in HCV-positive patients.

INTRODUCTION

Hepatitis C virus (HCV) infection remains a significant public health concern in Pakistan, where it is a leading cause of chronic liver disease, including cirrhosis and hepatocellular carcinoma (HCC). HCV is primarily transmitted through blood-to-blood contact, with common modes of transmission including the use of contaminated needles, unsterilized tattooing and body piercing equipment, drug use, and exposure to unscreened blood or blood products. Other transmission routes include sexual contact, perinatal exposure, and household contact with infected individuals. In particular, HCV is highly prevalent among hemodialysis patients due to the frequency of blood transfusions and prolonged dialysis duration (1, 2). The interspousal transmission of HCV is particularly notable in Asian countries, where cultural practices contribute to the increased spread from male to female partners (3).

Globally, HCV is classified into seven distinct genotypes, each with various subtypes, and these are distributed differently across regions (4). Understanding the genotype of HCV is crucial for guiding treatment, as different genotypes respond differently to antiviral therapies. For instance, patients with genotypes 2 and 3 exhibit greater sensitivity to combination therapy with alpha interferon and ribavirin compared to those with genotype 1, which is more resistant and requires longer treatment durations (5, 6). The burden of HCV infection in Pakistan is particularly alarming, with an estimated 12 million individuals affected, making it the second-highest global burden of the disease (7).Despite this high prevalence, more than 90% of HCV-infected individuals in Pakistan remain unaware of their condition, highlighting the urgent need for improved screening and awareness programs (8).

The thyroid gland, a critical component of the endocrine system, plays a pivotal role in regulating metabolic processes through the production of thyroid hormones, including thyroxine (T4) and triiodothyronine (T3). These hormones are crucial for maintaining the basal metabolic rate of all cells, including hepatocytes, which underscores the interdependence between thyroid function and liver health. The liver is integral to thyroid hormone metabolism, contributing to hormone conjugation, circulation, and deiodination processes (9). Dysregulation in thyroid function, particularly in patients with chronic liver diseases such as HCV infection, has been documented, with alterations in thyroid hormone levels often reflecting the severity of hepatic dysfunction. Specifically, reductions in total and free triiodothyronine (TT3 and FT3) levels are commonly observed and are associated with adaptive hypothyroid states that aim to conserve liver function and protein stores in response to liver impairment (10, 11).

Emerging evidence suggests that HCV infection is linked to various autoimmune disorders, including thyroid dysfunction, which can manifest as either hypothyroidism or hyperthyroidism. The prevalence of thyroid disorders, particularly hypothyroidism, is higher in individuals with chronic HCV infection compared to the general population

(12-14). Given the significant impact of thyroid dysfunction on the quality of life and clinical outcomes in HCV patients, understanding this association is critical for optimizing patient management. Therefore, this study was designed to evaluate the thyroid profile in HCV-positive patients, aiming to elucidate the potential correlation between HCV infection and thyroid hormone abnormalities.

MATERIAL AND METHODS

The study was a cross-sectional analysis conducted to evaluate the thyroid profile in patients infected with the hepatitis C virus (HCV). A total of 120 HCV-positive patients were enrolled in this study through a non-probability sampling technique. The participants included both male and female patients aged between 18 and 70 years who had tested positive for HCV. Patients who had been diagnosed with liver cirrhosis, those with a history of thyroid disorders, and individuals on medications known to affect thyroid function—such as carbamazepine, phenobarbitone, phenytoin, salicylates, and nonsteroidal anti-inflammatory drugs—were excluded from the study. All participants provided informed consent before being included in the study, and their confidentiality was maintained throughout the research process in accordance with the principles outlined in the Declaration of Helsinki (1).

Data were collected using a standardized questionnaire that recorded demographic information, HCV infection status, and relevant medical history. Approximately 3 milliliters of venous blood were drawn from each patient using a sterile technique and collected into clotted vacutainers. The samples were then centrifuged at 3000 rpm for ten minutes to separate the serum, which was subsequently used for the analysis of thyroid hormones. The levels of thyroid-stimulating hormone (TSH), free triiodothyronine (FT3), and free thyroxine (FT4) were measured using an automated immunoassay system (Abbott i1000sr), ensuring high sensitivity and specificity in the detection of thyroid function abnormalities.

The study's data were analyzed using IBM SPSS software, version 25.0. Descriptive statistics were utilized to summarize the demographic characteristics of the participants, with mean and standard deviation calculated for continuous variables and frequencies and percentages for categorical variables. The association between HCV infection and thyroid hormone levels was assessed using a one-sample t-test, with a p-value of less than 0.05 considered statistically significant. The results were interpreted in the context of existing literature to draw relevant conclusions about the relationship between HCV infection and thyroid dysfunction. The study was conducted with full ethical approval obtained from the relevant institutional review board, ensuring adherence to ethical standards in research involving human subjects. There were no conflicts of interest or funding sources to declare in relation to this study.

RESULTS

A total of 120 patients who tested positive for hepatitis C virus (HCV) were included in the study. The demographic characteristics of the participants are summarized in Table 1. Among the study population, 62 patients (51.66%) were female, and 58 patients (48.33%) were male. The mean age of the participants was 46.15 ± 14.25 years, with the majority of patients (63.34%) residing in rural areas. Most of the participants (70.83%) had been diagnosed with HCV for less than three months.

Table 1: Demographic Characteristics of HCV-Positive Patients

Variable	Frequency (%)
Gender	
Male	58 (48.33%)
Female	62 (51.66%)
Age Groups (Years)	
18-30	18 (15.0%)
31-50	58 (48.33%)
51-85	44 (36.66%)
Place of Living	
Rural	76 (63.34%)
Urban	44 (36.66%)
Duration of HCV Positivity	
<3 months	85 (70.83%)
>3 months	35 (29.17%)

The study assessed the thyroid hormone profile, including TSH, FT3, and FT4 levels, among the HCV-positive patients. The mean values for TSH, FT3, and FT4 were 1.15 ± 0.515 µIU/mL, 1.10 ± 0.351 pg/mL, and 1.12 ± 0.361 ng/dL, respectively. The distribution of thyroid hormone levels is detailed in Table 2. The results indicated that the majority of the HCV-positive patients had normal thyroid hormone levels, with 80.83% having normal TSH, 85.0% having normal FT3, and 86.66% having normal FT4 levels. However, a notable proportion of patients exhibited thyroid dysfunction, with 17.50% having elevated TSH levels and 11.66% and 10.83% having elevated FT3 and FT4 levels, respectively. The statistical analysis revealed a significant association between HCV infection and abnormal thyroid hormone levels, with p-values for TSH, FT3, and FT4 all below 0.05, indicating statistical significance.

These findings suggest a strong correlation between HCV infection and the presence of thyroid dysfunction,

emphasizing the need for regular thyroid function monitoring in HCV-positive patients to ensure early detection and management of potential thyroid-related complications.

Table 2: Thyroid Hormone Levels in HCV-Positive Patients

Thyroid Hormones	Frequency (%)	t-test (p-value)
TSH (0.35 - 4.94 µIU/mL)		
Normal	97 (80.83%)	0.001*
High	21 (17.50%)	
Low	2 (1.66%)	
FT3 (1.71 - 3.72 pg/mL)		
Normal	102 (85.0%)	0.001*
High	14 (11.66%)	
Low	4 (3.33%)	
FT4 (0.70 - 1.48 ng/dL)		
Normal	104 (86.66%)	0.008*
High	13 (10.83%)	
Low	3 (2.5%)	

*p-value considered statistically significant.

DISCUSSION

The present study explored the association between hepatitis C virus (HCV) infection and thyroid dysfunction, revealing a significant correlation between the two. The findings showed that a considerable proportion of HCV-positive patients exhibited abnormal thyroid hormone levels, particularly elevated TSH, FT3, and FT4 levels. These results are consistent with previous studies that have documented an increased prevalence of thyroid disorders among individuals with chronic HCV infection, highlighting the virus's potential role in disrupting thyroid function (15, 16-21).

The study's findings align with the work of Nazary et al., who observed a higher prevalence of both overt and subclinical hypothyroidism in HCV-positive patients compared to a control group (15). Similarly, Gupta et al. found significant differences in thyroid hormone levels between HCV-infected individuals and healthy controls, further supporting the notion that HCV infection may contribute to thyroid dysfunction (21). The present study also corroborated the observations of Khan et al., who reported a predominance of female patients with thyroid abnormalities in the context of HCV infection, reflecting the gender distribution seen in this study (16).

The liver plays a crucial role in the metabolism of thyroid hormones, and its dysfunction due to HCV infection may impair the normal regulation of these hormones. The significant association found between abnormal thyroid hormone levels and HCV infection in this study suggests that the virus may directly or indirectly influence thyroid function, possibly through mechanisms involving immune-mediated damage or alterations in hepatic deiodinase activity, which is essential for thyroid hormone conversion (19, 20). The finding that a significant number of patients had elevated FT3 and FT4 levels may indicate an adaptive response of the thyroid gland to maintain metabolic homeostasis in the presence of liver dysfunction caused by HCV (13).

One of the strengths of this study was its focus on a specific population of HCV-positive patients without liver cirrhosis, allowing for a more precise evaluation of the association between HCV and thyroid dysfunction without the confounding effects of advanced liver disease. However, the cross-sectional design of the study limited the ability to establish a causal relationship between HCV infection and thyroid abnormalities. Longitudinal studies are needed to clarify the temporal relationship between these conditions and to explore the underlying mechanisms further.

Another limitation of the study was the relatively small sample size, which may have affected the generalizability of the findings. Larger, multicenter studies would be valuable to confirm these results and to provide a more comprehensive understanding of the prevalence and impact of thyroid dysfunction in HCV-positive populations. Additionally, the study did not assess other factors that could influence thyroid function, such as the use of antiviral therapy, which has been shown to affect thyroid hormone levels in some patients (22, 23).

Despite these limitations, the study highlights the importance of regular monitoring of thyroid function in patients with HCV infection. Early detection of thyroid abnormalities in this population is crucial for preventing the potential complications associated with untreated thyroid dysfunction, such as cardiovascular disease, metabolic disturbances, and impaired quality of life. Clinicians should consider incorporating routine thyroid function tests into the management plan for HCV-positive patients, particularly those with risk factors for thyroid disease.

Conclusion

In conclusion, the study demonstrated a significant association between HCV infection and thyroid dysfunction, reinforcing the need for vigilant screening and management of thyroid health in this population. Future research should focus on elucidating the mechanisms underlying this association and on developing targeted strategies to mitigate the impact of thyroid disorders in HCV-infected individuals.

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