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Research Article

Study of Thyroid and Insulin Dysfunction in Hepatitis B and Hepatitis C Patients

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Abstract

Chronic Hepatitis B virus (HBV) and Hepatitis C virus (HCV) infections are associated with several extrahepatic manifestations, including endocrine and metabolic abnormalities. Thyroid dysfunction and insulin resistance have been increasingly recognized as important complications that may adversely affect disease progression and patient outcomes. This study evaluated thyroid dysfunction and insulin resistance among patients with chronic Hepatitis B and Hepatitis C infections.

Material and Methods: This hospital-based observational study was conducted in the Department of Medicine, Muzaffarnagar Medical College, Uttar Pradesh, over 18 months. A total of 100 patients aged 18–65 years with chronic Hepatitis B or Hepatitis C infection were included. Detailed clinical evaluation and laboratory investigations were performed, including liver function tests, thyroid profile (FT3, FT4, TSH), fasting blood glucose, fasting insulin, HbA1c, and HOMA-IR. Data were analysed using SPSS version 27.0.

Results: Among the 100 participants, 57% had Hepatitis C and 43% had Hepatitis B infection. Thyroid dysfunction was observed in 45.61% of Hepatitis C patients and 41.86% of Hepatitis B patients, with subclinical hypothyroidism being the most common abnormality (26.32% and 25.58%, respectively). Mean TSH levels were higher in Hepatitis C patients (4.14±1.92 µIU/mL) compared to Hepatitis B patients (3.52±1.75 µIU/mL). Hepatitis C patients also exhibited higher fasting insulin levels (13.65±2.50 vs. 11.91±2.04 µIU/mL), fasting glucose levels (101.00±6.38 vs. 97.74±5.63 mg/dL), and HbA1c levels (6.17±0.45% vs. 5.87±0.32%). Insulin resistance was present in 35.09% of Hepatitis C patients and 34.88% of Hepatitis B patients. Liver function abnormalities were more pronounced among patients with Hepatitis C.

Conclusion: Thyroid dysfunction and insulin resistance are common extrahepatic manifestations of chronic viral hepatitis, with Hepatitis C showing greater endocrine and metabolic derangements than Hepatitis B. Routine assessment of thyroid and glycemic parameters may facilitate early detection and management of these complications.

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KEYWORDS: Hepatitis B, Hepatitis C, Thyroid Dysfunction, Insulin Resistance, HOMA-IR.

1. INTRODUCTION

Chronic Hepatitis B virus (HBV) and Hepatitis C virus (HCV) infections are still a major health problem that affects millions of people globally and are a major cause of chronic liver disease, cirrhosis, and hepatocellular carcinoma [1]. These viral infections have been known to have hepatic effects, but are also being recognized as systemic diseases with a number of extrahepatic effects [2]. Of these, endocrine and metabolic disorders have become a focus of interest because of their role in disease activity, treatment response and quality of life [3]. HBV and HCV infection load is particularly high in developing countries, where the lack of early diagnosis and extended disease course aggravates the risk of metabolic complications [4].

The liver is a major site for the metabolism, transport, and regulation of thyroid hormones. Therefore, chronic liver damage could lead to disturbances in the hypothalamic–pituitary–thyroid axis and thyroid hormone homoeostasis [5]. Additionally, chronic viral infections and immune dysfunction have been associated with thyroid dysfunction such as hypothyroidism, hyperthyroidism, and subclinical thyroid dysfunction [6]. Thyroid disorders have been reported to be more common in patients with chronic viral hepatitis, especially in patients with HCV infection, and the interaction between viral persistence, immune activation and endocrine function is complex [7,8].

Another important extrahepatic manifestation of chronic viral hepatitis is insulin resistance. HCV has been known to disrupt insulin signaling pathways via inflammatory cytokine mediated mechanisms, leading to impaired glucose metabolism and type 2 diabetes mellitus [9]. The association is more common in HCV infection but chronic HBV infection may also be associated with metabolic disturbances secondary to chronic inflammation and liver dysfunction [10]. Thyroid dysfunction and insulin resistance may interact to exacerbate metabolic disturbances, leading to hepatic steatosis, progression of fibrosis, cardiovascular manifestations, and poor outcomes [11]. Although there is increasing information on the endocrine dysfunction in chronic viral hepatitis, there is limited information on the differences between thyroid and insulin dysfunction in chronic Hepatitis B and Hepatitis C, especially in Indian population. This understanding of the metabolic changes may help enable early detection, specific monitoring, and patient management. Thus, the aim of the present study was to determine the prevalence of thyroid dysfunction and insulin resistance in chronic Hepatitis B and C infected patients and to determine the effect of chronic viral hepatitis on thyroid hormone profile and glycemic status.

2. MATERIALS AND METHODS

This is an observational study done in the Department of Medicine, Muzaffarnagar Medical College, Muzaffarnagar, Uttar Pradesh for 18 months. A total of 12 months was spent on the recruitment of the patients and collection of the data, and 6 months was spent compiling, analysing and interpreting the data. After obtaining written informed consent, 100 patients with chronic Hepatitis B or chronic Hepatitis C infection were enrolled in the study. Before the study was started, an ethical clearance was obtained from the Institutional Ethics Committee. The study population consisted of adult patients (age 18–65 years), of both sexes with well-established chronic liver disease due to chronic Hepatitis B or Hepatitis C infection. Chronic liver disease was diagnosed by clinical assessment and ultrasonographic features suggestive of chronic hepatic involvement such as changes in the liver size and echogenicity. Excluded were patients who did not give consent, who had chronic kidney disease, nephrotic syndrome, hepatocellular carcinoma, organ failure, malignancy, active systemic infections, or a history of radiotherapy or chemotherapy. Those taking drugs that affect thyroid function such as levothyroxine, propylthiouracil, carbimazole, iodine-containing drugs, amiodarone and beta-blockers were also excluded.

Detailed clinical evaluation of each participant was done based on thorough general and systemic examination and detailed history taking. Demographic data, clinical observations and pertinent medical history were documented on a predesigned case record form. Laboratory investigations comprised of viral marker assessment was performed using enzyme-linked immunosorbent assay (ELISA) for hepatitis B surface antigen (HBsAg), anti-HCV antibody and HIV. Liver function tests were such as estimation of total bilirubin, direct bilirubin, indirect bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), serum total protein and albumin. Serum urea and creatinine levels were used to determine renal function and serum sodium and potassium levels were measured as part of the electrolyte profile.

Thyroid function was assessed by estimation of serum free triiodothyronine (FT3), free thyroxine (FT4) and thyroid-stimulating hormone (TSH) by electrochemiluminescence immunoassay technique. The reference ranges used were 2.1–4.4 pg/mL for FT3, 0.8–2.7 ng/dL for FT4, and 0.35–5.5 μIU/mL for TSH. Insulin dysfunction was assessed by measuring fasting blood glucose, fasting serum insulin, glycated hemoglobin (HbA1c), and calculating Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) (fasting glucose × fasting insulin/22.5).

All the data collected were coded to ensure confidentiality and analyzed using Statistical Package for the Social Sciences (SPSS) version 27.0. The quantitative variables were expressed as mean ± standard deviation (SD) and quantitative variables were compared using Student's t test, and qualitative variables were analyzed using chi square test. A p-value <0.05 was considered statistically significant.

3. RESULTS

The study included 100 patients diagnosed with chronic viral hepatitis. The majority of participants belonged to the 31–40 years age group (41.0%), followed by 51–60 years (25.0%) and 41–50 years (24.0%). Only 10.0% of patients were aged between 21 and 30 years. Males constituted 63.0% of the study population, while females accounted for 37.0%. With respect to viral etiology, 57.0% of patients had Hepatitis C infection and 43.0% had Hepatitis B infection. (Table 1)

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants

Variable	Category	Total (N=100)
Age group	21–30 years	10 (10.0%)
	31–40 years	41 (41.0%)
	41–50 years	24 (24.0%)
	51–60 years	25 (25.0%)
Gender	Male	63 (63.0%)
	Female	37 (37.0%)
Hepatitis	Hepatitis C	57 (57.0%)
	Hepatitis B	43 (43.0%)

Comparison of liver function parameters demonstrated greater biochemical derangement among patients with Hepatitis C than those with Hepatitis B. Mean total bilirubin, direct bilirubin, and indirect bilirubin levels were higher in Hepatitis C patients (1.54 ± 0.46 mg/dL, 0.55 ± 0.24 mg/dL, and 0.99 ± 0.22 mg/dL, respectively) compared to Hepatitis B patients (1.21 ± 0.32 mg/dL, 0.38 ± 0.17 mg/dL, and 0.83 ± 0.16 mg/dL, respectively). Similarly, AST and ALT levels were elevated in the Hepatitis C group (57.89 ± 12.68 U/L and 63.77 ± 15.18 U/L) relative to the Hepatitis B group (48.84 ± 10.00 U/L and 53.14 ± 12.08 U/L). In contrast, total protein and albumin levels were lower among Hepatitis C patients, indicating relatively impaired hepatic synthetic function. (Table 2)

Table 2: Comparison of Liver Function Parameters Between Hepatitis C and Hepatitis B Patients

Parameter	Hepatitis C (n=57) Mean ± SD	Hepatitis B (n=43) Mean ± SD
Total Bilirubin (mg/dL)	1.54 ± 0.46	1.21 ± 0.32
Direct Bilirubin (mg/dL)	0.55 ± 0.24	0.38 ± 0.17
Indirect Bilirubin (mg/dL)	0.99 ± 0.22	0.83 ± 0.16
AST (U/L)	57.89 ± 12.68	48.84 ± 10.00
ALT (U/L)	63.77 ± 15.18	53.14 ± 12.08
Total Protein (g/dL)	6.16 ± 0.44	6.46 ± 0.34
Albumin (g/dL)	3.15 ± 0.42	3.43 ± 0.29

Assessment of thyroid status revealed that euthyroidism was the most common thyroid state in both groups, observed in 54.4% of Hepatitis C patients and 58.1% of Hepatitis B patients. Subclinical hypothyroidism was the most frequent thyroid dysfunction, affecting 26.3% of Hepatitis C patients and 25.6% of Hepatitis B patients. Overt hypothyroidism was present in 14.0% and 11.6% of Hepatitis C and Hepatitis B patients, respectively, whereas hyperthyroidism was relatively uncommon, occurring in 5.3% and 4.7% of patients, respectively. (Table 3)

Table 3: Thyroid Function Status Among Hepatitis C and Hepatitis B Patients

Thyroid Status	Hepatitis C (n=57)	Hepatitis B (n=43)
Hyperthyroid	3 (5.3%)	2 (4.7%)
Hypothyroid	8 (14.0%)	5 (11.6%)
Subclinical Hypothyroid	15 (26.3%)	11 (25.6%)
Euthyroid	31 (54.4%)	25 (58.1%)

Analysis of thyroid hormone profiles showed lower mean FT3 and FT4 levels among Hepatitis C patients compared to Hepatitis B patients. The mean FT3 level was 2.87 ± 0.84 pg/mL in Hepatitis C and 3.22 ± 0.75 pg/mL in Hepatitis B patients, while the corresponding FT4 values were 1.26 ± 0.58 ng/dL and 1.44 ± 0.50 ng/dL. Conversely, TSH levels were higher in the Hepatitis C group (4.14 ± 1.92 µIU/mL) than in the Hepatitis B group (3.52 ± 1.75 µIU/mL), suggesting a greater tendency toward thyroid dysfunction among patients with Hepatitis C infection. (Table 4)

Table 4: Comparison of Thyroid Hormone Profile Between Hepatitis C and Hepatitis B Patients

Parameter	Hepatitis C Mean ± SD	Hepatitis B Mean ± SD
FT3 (pg/mL)	2.87 ± 0.84	3.22 ± 0.75
FT4 (ng/dL)	1.26 ± 0.58	1.44 ± 0.50
TSH (µIU/mL)	4.14 ± 1.92	3.52 ± 1.75

Evaluation of glycemic and insulin resistance parameters demonstrated higher fasting insulin, fasting glucose, and HbA1c levels in patients with Hepatitis C than in those with Hepatitis B. Mean fasting insulin levels were 13.65 ± 2.50 µIU/mL in Hepatitis C patients compared to 11.91 ± 2.04 µIU/mL in Hepatitis B patients. Similarly, fasting glucose levels were 101.00 ± 6.38 mg/dL and 97.74 ± 5.63 mg/dL, while HbA1c levels were 6.17 ± 0.45% and 5.87 ± 0.32%, respectively. However, mean HOMA-IR values were comparable between the two groups, measuring 2.52 ± 0.99 in Hepatitis C patients and 2.54 ± 0.80 in Hepatitis B patients. (Table 5)

Table 5: Comparison of Glycemic and Insulin Resistance Parameters Between Hepatitis C and Hepatitis B Patients

Parameter	Hepatitis C Mean ± SD	Hepatitis B Mean ± SD
Fasting Insulin (µIU/mL)	13.65 ± 2.50	11.91 ± 2.04
Fasting Glucose (mg/dL)	101.00 ± 6.38	97.74 ± 5.63
HbA1c (%)	6.17 ± 0.45	5.87 ± 0.32
HOMA-IR	2.52 ± 0.99	2.54 ± 0.80

Insulin resistance was identified in approximately one-third of patients in both groups. Among Hepatitis C patients, 20 individuals (35.1%) demonstrated insulin resistance, whereas 15 patients (34.9%) with Hepatitis B had insulin resistance. The remaining 64.9% of Hepatitis C patients and 65.1% of Hepatitis B patients did not exhibit insulin resistance, indicating a similar prevalence of insulin resistance in both viral hepatitis groups. (Table 6)

Table 6: Distribution of Insulin Resistance Among Hepatitis C and Hepatitis B Patients

Insulin Resistance	Hepatitis C (n=57)	Hepatitis B (n=43)
Present	20 (35.1%)	15 (34.9%)
Absent	37 (64.9%)	28 (65.1%)

Comparison of renal function and electrolyte parameters showed modest differences between the two groups. Mean serum sodium levels were slightly lower in Hepatitis C patients (136.33 ± 2.33 mEq/L) than in Hepatitis B patients (137.65 ± 1.79 mEq/L). Serum potassium levels were marginally higher in the Hepatitis C group (4.55 ± 0.34 mEq/L) compared to the Hepatitis B group (4.30 ± 0.26 mEq/L). Likewise, mean serum urea and creatinine levels were higher among Hepatitis C patients (36.44 ± 5.90 mg/dL and 1.13 ± 0.19 mg/dL, respectively) than among Hepatitis B patients (32.40 ± 4.08 mg/dL and 1.01 ± 0.13 mg/dL, respectively). (Table 7)

Table 7: Comparison of Renal Function and Electrolyte Parameters Between Hepatitis C and Hepatitis B Patients

Parameter	Hepatitis C Mean ± SD	Hepatitis B Mean ± SD
Sodium (mEq/L)	136.33 ± 2.33	137.65 ± 1.79
Potassium (mEq/L)	4.55 ± 0.34	4.30 ± 0.26
Urea (mg/dL)	36.44 ± 5.90	32.40 ± 4.08
Creatinine (mg/dL)	1.13 ± 0.19	1.01 ± 0.13

4. DISCUSSION

Presently, thyroid dysfunction and insulin resistance in patients with chronic Hepatitis B and Hepatitis C infection were assessed and it was observed that endocrine and metabolic abnormalities were not uncommon in both the groups with relatively higher frequency in Hepatitis C group. Most of the study participants were in the age group 31-40 years (41%) and male population accounted for 63% of the study population.

This is similar to what has been reported in previous studies. The mean age of the chronic hepatitis patients with thyroid dysfunction was 41±10 years as reported by Nazary et al., and 46.15±14.25 years for the HCV-infected patients as reported by Ali et al. [12,13]. Kaya et al. also reported a mean age of 38.96±11.57 years with male predominance of 60.84% which is similar to the current study [14]. The predominance of middle-aged adults may be explained by the long-term persistence of the virus, the cumulative liver damage and the ongoing inflammatory response, which could cause endocrine and metabolic disturbances over time.

Frequent thyroid dysfunction was seen in both viral hepatitis groups. Thyroid dysfunction was found in 41.86% of Hepatitis B patients, the most common being subclinical hypothyroidism (25.58%), followed by overt hypothyroidism (11.63%) and hyperthyroidism (4.65%). Likewise, 45.61% of the Hepatitis C patients were detected to be having thyroid dysfunction, 26.32% of which were subclinical hypothyroidism, 14.04% overt hypothyroidism and 5.26% hyperthyroidism. These rates were significantly higher than those reported by Antonelli et al. (6.2% hypothyroidism and 8.5% subclinical hypothyroidism) and Liu et al. (10.7% subclinical hypothyroidism and 12.1% hyperthyroidism) in HBV patients [15,16]. Nazary et al. found hypothyroidism and subclinical hypothyroidism in 10.6% and 6.0% of patients with HCV infection, respectively, while Shen et al. reported pooled prevalence rates of 12.8% and 9.3%, respectively [12,17]. In addition, the current study showed that the mean TSH level among Hepatitis C patients (4.14±1.92 µIU/mL) was higher than that of Hepatitis B patients (3.52±1.75 µIU/mL) with lower levels of FT3 and FT4. Nazary et al. reported similar rise in TSH and Ali et al. reported abnormal TSH, FT3 and FT4 levels in 17.5%, 11.66% and 10.83% of HCV-positive patients respectively [12,13]. These findings contribute to the increasing evidence of immune-mediated and metabolic effects of chronic viral hepatitis, especially HCV infection, on thyroid function.

Another major finding in the present study was insulin resistance. The prevalence of insulin resistance was 34.88% in Hepatitis B patients and 35.09% in Hepatitis C patients. Patients with Hepatitis C demonstrated higher fasting insulin levels (13.65±2.50 µIU/mL), fasting glucose levels (101.00±6.38 mg/dL), and HbA1c levels (6.17±0.45%) compared with Hepatitis B patients (11.91±2.04 µIU/mL, 97.74±5.63 mg/dL, and 5.87±0.32%, respectively). These results are similar to those of Memon et al. and Mehta et al. who reported insulin resistance or diabetes in 31.5% and 38.0% of HCV patients, respectively [18,19]. Likewise, Cai et al. reported diabetes mellitus in 22.1% of the HBV patients, while

Wang et al. reported 19.4% [20]. HBV patients had higher fasting insulin (23.63%) and HOMA-IR (47.27%) levels than HCV patients who had 68.8% and 77.7% respectively [21]. The increased insulin and glycemic index levels in the current study in HCV patients is in line with the hypothesis of HCV-induced insulin resistance due to chronic inflammation, impaired insulin signalling pathways by cytokines and hepatic steatosis.

Biochemical evaluation of liver function showed that there was higher biochemical impairment in HCVs. Mean total bilirubin, AST, and ALT levels were higher in Hepatitis C patients (1.54±0.46 mg/dL, 57.89±12.68 U/L, and 63.77±15.18 U/L, respectively) than in Hepatitis B patients (1.21±0.32 mg/dL, 48.84±10.00 U/L, and 53.14±12.08 U/L, respectively). Moreover, the serum albumin level was also reduced in the Hepatitis C group (3.15±0.42 g/dL compared with 3.43±0.29 g/dL), indicating impaired hepatic synthetic function. These results are consistent with the results of Kaya et al. who found that the prevalence of insulin resistance and metabolic abnormalities increased with the deterioration of liver function parameters in chronic hepatitis patients [14]. Mildly higher serum urea (36.44±5.90 mg/dL vs. 32.40±4.08 mg/dL) and creatinine levels (1.13±0.19 mg/dL vs. 1.01±0.13 mg/dL) were also observed among Hepatitis C patients, whereas electrolyte levels remained largely within normal limits in both groups. All of these suggest that chronic Hepatitis C infection is linked to greater hepatic, endocrine and metabolic abnormalities than Hepatitis B infection, highlighting the systemic nature of chronic viral hepatitis.

The current study has some limitations. The study was performed in one tertiary care centre with a relatively small sample size and thus the results may not be generalizable to the general population. The cross-sectional observational study prevented the evaluation of causal relationship between chronic viral hepatitis and endocrine dysfunction. However, thyroid autoantibodies, inflammatory cytokines and viral load measurements were not assessed, thus restricting further analysis of the mechanisms of thyroid dysfunction and insulin resistance. In addition, no long-term follow-up was done to determine the course of endocrine and metabolic abnormalities over time.

5. CONCLUSION

The present study demonstrated that thyroid dysfunction and insulin resistance are common extrahepatic manifestations among patients with chronic Hepatitis B and Hepatitis C infections. Subclinical hypothyroidism emerged as the most frequent thyroid abnormality in both groups, while Hepatitis C patients exhibited comparatively higher TSH levels, greater disturbances in thyroid hormone profile, higher fasting insulin, fasting glucose, and HbA1c levels, along with more pronounced liver function abnormalities. Insulin resistance was observed in approximately one-third of patients with both infections. These findings highlight the close interplay between chronic viral hepatitis and endocrine-metabolic dysfunction and support the need for routine assessment of thyroid function and glycemic status in patients with chronic Hepatitis B and Hepatitis C to facilitate early detection and appropriate management of these complications.

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