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Vitamin D Deficiency and Its Association with Cirrhosis among Patients with Chronic Hepatitis C Infection

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ABSTRACT

Vitamin D deficiency represents a common metabolic disturbance in chronic liver disorders, with a particularly high prevalence among individuals affected by chronic hepatitis C virus (HCV) infection. Evidence reported that progressive hepatic dysfunction can be linked to the waning levels of vitamin D; the information on South Asian populations is scarce. **Objectives:** To evaluate the association between deficient serum vitamin D levels and the presence of liver cirrhosis among individuals with HCV infection. **Methods:** The case-control study involved 162 chronic hepatitis C patients in the Department of Medicine, Kishwar Fazal Teaching Hospital, Lahore. Demographic, clinical, and laboratory parameters were taken. The level of vitamin D in serum was determined and categorized based on the severity. The statistical analysis was conducted using SPSS version 25.0 through suitable tests. **Results:** The study population had a vitamin D deficiency of 92.0%. Cases of severe vitamin D deficiency were found 30.2 higher in cirrhotic patients than in non-cirrhotic patients (30.2% vs. 14.0%). Severe vitamin D deficiency, female gender, and longer HCV infection were significantly related to cirrhosis. Extremely vitamin-D-deficient patients showed a high concentration of alanine aminotransferase and aspartate aminotransferase, elevated serum bilirubin, and decreased serum albumin, which is indicative of severe hepatic dysfunction. **Conclusions:** In chronic hepatitis C patients, vitamin D deficiency, especially the severe form, is highly correlated with cirrhosis and poor biochemical markers in the patients. These results justify the inclusion of regular screening of vitamin D status in the overall evaluation of patients with chronic HCV infection.

INTRODUCTION

Chronic hepatitis C virus (HCV) infection constitutes a substantial global public health challenge, affecting an estimated 50–58 million individuals worldwide, with marked regional variation in prevalence [1]. Persistent HCV infection induces ongoing hepatic inflammation and progressive fibrotic remodeling, culminating in liver cirrhosis in approximately 15–30% of untreated patients over a period of two to three decades [2]. HCV cirrhosis is a key cause of mortality associated with the liver and one of the most common liver transplant indications in most countries around the world. Cirrhosis has clinical effects such as portal hypertension, ascites, hepatic encephalopathy, and hepatocellular carcinoma, which greatly affect the quality of life and survival [3]. The prevalence of HCV in South Asia is between 2-5%, and cirrhosis is quite often detected at the first clinical examination. Even though direct-acting antivirals have increased the rate of viral eradication, pre-existing cirrhosis is usually not eliminated despite the elimination of the virus [4]. Vitamin D deficiency is also highly prevalent at

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a global level, impacting more than one billion people, with disproportionately higher rates observed among individuals with chronic liver disease [5]. Among individuals with HCV, studies have documented a broad variation in the prevalence of inadequate vitamin D levels, ranging from approximately 46% to 90%, with differences largely attributable to geographic location and the extent of liver disease [6]. Vitamin D requires the liver to be metabolized via 25-hydroxylation, and hepatic impairment has a direct negative impact on the process in cirrhotic patients [7]. Food deficiency, poor intestinal absorption, fewer days in the sun, and systemic inflammation are other factors that cause low vitamin D levels in advanced liver disease [8]. Observational research continues to reveal a pronounced reduction in the level of serum 25-hydroxyvitamin D in cirrhotic patients as compared to those who are non-cirrhotic but infected with HCV. The degree of vitamin D deficiency has been reported to be associated with the increased Child-Pugh class and Model of End-Stage Liver Disease score [9]. Although there is accumulating evidence, there is limited data on vitamin D-deficient patients having cirrhosis among patients with chronic HCV infection, especially in those resource-limited environments [10]. The numerous existing studies are based on mixed population groups and have different definitions of vitamin D deficiency and cirrhosis; as a result, comparing the findings with each other is not easy [11]. Also, such confounding factors as nutritional status, exposure to antiviral treatment, presence of sunshine, and comorbid liver diseases are frequently poorly controlled [12]. Locally produced data of South Asian populations is stereotyped, and both HCV infection and lack of vitamin D are extremely common [13].

A specific Pakistani investigation has never focused on the relationship between cirrhosis and vitamin D deficiency in chronic HCV patients, who are a specific group of subjects under a case-control design. The current regional studies are also restricted to mixed etiologies or not stratified by the severity of deficiencies. Such disparity prevents evidence-based screening and supplementation practices in the local high-burden locations. The present study offers specific information about the association between vitamin D-cirrhosis and the HCV population. This association is crucial to the proper risk stratification and monitoring of diseases, and it is important to understand this association in particular clinical settings. Furthermore, vitamin D is a potentially alterable factor that would be amenable to the low-cost interventions provided there is a serious association. This research aims to evaluate the association between deficient serum vitamin D levels and the presence of liver cirrhosis among individuals with HCV infection.

METHODS

A case control study was carried out at the Department of Medicine, Kishwar Fazal Teaching Hospital, Lahore, i.e., an attached tertiary care facility under Amna Inayat Medical College, from July 2025 to December 2025. The ethical approval of the research was obtained from the Institutional Review Board of Amna Inayat Medical College (AIMEC), Lahore (Ref/10/RE). Before inclusion in the study, participants gave written informed consent after being adequately informed about the purpose and conduct of the research. Sample size calculation was conducted in accordance with the World Health Organization's recommended calculator. The study consisted of 162 patients (81 patients in each group). It was calculated at 5% level of significance, 80% power of the study, and an expected prevalence of vitamin D deficiency among the HCV patients with cirrhosis, and 14.0% among the HCV patients without cirrhosis. The non-probability consecutive sampling was used. The eligible patients who reported to the medical outpatient department (OPD) within the study period were recruited consecutively until the required sample size was reached. Included were patients aged between 16 and 65 years of any gender with a successful diagnosis of HCV infection and a treatment history of over three months. The cases were considered as HCV patients diagnosed with cirrhosis, and the controls were HCV patients without cirrhosis. Patients who reported taking vitamin D or calcium supplements were excluded. Individuals with cirrhosis due to other etiological causes, such as hepatitis B virus infection or other causes, were also excluded. Pre-enrollment demographic and clinical data were collected with a pre-tested proforma. Vitamin D in serum was measured at the hospital pathology lab and recorded on lab reports. The level of vitamin D below 20 ng/mL was considered vitamin D deficiency.

The Statistical Package of Social Sciences (SPSS) version 25.0 was used to perform data entry and data analysis. The Shapiro-Wilk test was used to determine whether the quantitative variables were normal. Constant and categorical variables were described as mean ± standard deviation and frequencies and percentages, respectively. Stratification was used to control potential confounders, including age, gender, BMI, years of infection, residence, diabetes, hypertension, smoking, anemia, family history of HCV infection, and substance abuse. The chi-square test was used to make post-stratification comparisons of vitamin D deficiency arguments, with a p-value of less than 0.05 regarded as statistically significant.

RESULTS

Patients in the cirrhosis group were older (mean age of 52.4 years) as compared to the non-cirrhotic group (mean age of 45.9 years), and they were more likely to be aged over 55

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years. above 55 years (35.8% versus 22.2%) (Table 1).

Table 1: Demographics of Study Participants

Variables	HCV with Cirrhosis (81 Patients)	HCV without Cirrhosis (81 Patients)
Mean Age (years)	52.4 ± 8.6	45.9 ± 9.2
Age >55 Years	29 (35.8%)	18 (22.2%)
Male	49 (60.5%)	46 (56.8%)
Female	32 (39.5%)	35 (43.2%)
Mean BMI (kg/m^2)	26.8 ± 4.1	27.3 ± 4.5
BMI ≥30 kg/m^2	21 (25.9%)	24 (29.6%)
Urban Residence	47 (58.0%)	51 (63.0%)
Rural Residence	34 (42.0%)	30 (37.0%)

The patients with cirrhosis proved to have longer disease periods and greater loads of metabolic and hematologic complications. There were higher mean values of AST and ALT in the patients with prior cirrhosis, indicating severe liver damage. The derangement of coagulation was manifested by increased INR (1.46 vs 1.09), whereas hypoalbuminemia was significantly more intense in cirrhosis, which highlights the defective synthetic liver activity (Table 2).

Table 2: Biochemical Characteristics of Study Participants

Variables	HCV with Cirrhosis	HCV without Cirrhosis
Duration of HCV infection (years)	8.1 ± 3.2	5.4 ± 2.6
Diabetes mellitus	26 (32.1%)	23 (28.4%)
Hypertension	29 (35.8%)	21 (25.9%)
Anemia (Hb <10 g/dL)	31 (38.3%)	18 (22.2%)
Mean AST (IU/L)	78.6 ± 34.2	49.3 ± 21.7
Mean ALT (IU/L)	71.9 ± 29.8	55.4 ± 24.1
Mean INR	1.46 ± 0.31	1.09 ± 0.18
Mean Serum Albumin (g/dL)	2.9 ± 0.6	3.7 ± 0.5

Deficiency of vitamin D was widespread in both groups, and extreme deficiency was more pronounced among cirrhotic patients, with almost one-third of these patients being affected. Conversely, non-cirrhotic patients had a larger percentage of mild-to-moderate deficiency and a higher percentage of normal vitamin D, indicating that the depletion is progressive, and with increasing liver disease, the level of deficiency increases (Table 3).

Table 3: Vitamin D Status among Patients with HCV Infection

Vitamin D Category	Cirrhotic Group	Non-Cirrhotic Group
Normal Level	4 (4.7%)	9 (10.6%)
Mild Deficiency	13 (16.3%)	18 (22.8%)
Moderate Deficiency	40 (48.8%)	43 (52.6%)
Severe Deficiency	24 (30.2%)	11 (14.0%)

There was also a significant linkage of higher risks of severe deficiency of vitamin D with female gender and liver cirrhosis, respectively (p=0.019 and 0.014). Another important determinant was a longer hepatitis C infection (>7 years), with the affected patients showing an odds ratio of 2.31 (p=0.036), indicating that a longer period of exposure to HCV inflammation is one of the determinants of progressive vitamin D deficiency (Table 4).

Table 4: Association of Severe Vitamin D Deficiency with Laboratory Predictors

Variables	Severe Deficiency Present (n)	Severe Deficiency Absent (n)	Odds Ratio (95% CI)	p-value
Liver cirrhosis	24	57	2.58 (1.21–5.49)	0.014
Female gender	19	48	2.74 (1.18–6.33)	0.019
Diabetes mellitus	15	34	1.18 (0.55–2.54)	0.670
BMI ≥30 kg/m^2	11	34	0.89 (0.39–2.01)	0.780
Duration of HCV ≥7 Years	22	46	2.31 (1.06–5.03)	0.036

The mean serum vitamin D concentration in the severe group was 6.1 ± 0.9 ng/mL, and that of the non-severe group was 18.9 ± 5.8 ng/mL, giving a mean difference of -12.8 ng/mL (p<0.001), which validated the existence of a significant biochemical difference between the severe and non-severe groups. Mean AST levels were significantly higher in patients with severe deficiency (82.4 ± 36.7 IU/L) than in those without severe deficiency (52.6 ± 23.4 IU/L), with an absolute mean difference of 29.8 IU/L (p=0.001). On the same note, ALT was greater at 74.1 ± 31.5 IU/L compared to 56.3 ± 25.2 IU/L at a mean difference of +17.8 IU/L (p=0.014). Mean serum albumin showed significant differences between patients with severe vitamin D deficiency, having 2.8 > 0.5 g/dL as compared to patients with no severe deficiency, having 3.6 > 0.6 g/dL, giving a mean difference of -.8 g/dl (p<0.001). The severe deficiency group had accordingly higher levels of total bilirubin, accordingly: 2.4 ± 1.1 mg/dL and 1.3 ± 0.6 mg/dL, respectively, with a mean difference of +1.1 mg/dl (p=0.002) (Table 5).

Table 5: Comparison of Laboratory Parameters According to Vitamin D Status in HCV Infection

Laboratory Parameters	Severe Deficiency Group	Non-Severe / Normal Group	Mean Difference	p-value
Serum Vitamin D (ng/mL)	6.1 ± 0.9	18.9 ± 5.8	–12.8	<0.001
AST (IU/L)	82.4 ± 36.7	52.6 ± 23.4	+29.8	0.001
ALT (IU/L)	74.1 ± 31.5	56.3 ± 25.2	+17.8	0.014
INR	1.51 ± 0.33	1.12 ± 0.19	+0.39	<0.001
Serum Albumin (g/dL)	2.8 ± 0.5	3.6 ± 0.6	–0.8	<0.001
Total Bilirubin (mg/dL)	2.4 ± 1.1	1.3 ± 0.6	+1.1	0.002

DISCUSSION

This research aimed to evaluate the association between deficient serum vitamin D levels and the presence of liver cirrhosis among individuals with HCV infection. The high level of severe vitamin D deficiency was significantly greater in patients with HCV-induced cirrhosis, with a prevalence of 30.2% vs 14.0% in patients with cirrhosis and non-cirrhotic patients, respectively. The cirrhosis and severe vitamin D deficiency resulted statistically

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significant correlation with an odds ratio of 2.58 (p=0.014). Such a level of association is similar to the results by Barchetta *et al.* who have shown a twofold rise in severe vitamin D deficiency incidence among cirrhotic HCV individuals in a European cohort of 468 participants [14]. Besides, the prolonged time of HCV infection (more than 7 years) was independently linked to severe deficiency (p=0.036). Rezaei *et al.* also reported similar temporal relationships, indicating that the levels of vitamin D decreased by about 1.2 ng/mL annually during untreated chronic HCV infection [15]. In this research, the analysis also showed that women had much greater chances of severe deficiency of vitamin D, with an odds ratio of 2.74 (p=0.019). Compared to male patients, 29.7% of female patients were severely deficient, against 13.1%, which is a difference that is reflected by Middle Eastern and South Asian populations with cultural, behavioral, and hormonal differences that lead to reduced exposure to the sun and disturbed metabolism of vitamin D in women [16]. This association did not end following the consideration of BMI and diabetes in the current study, indicating that sex-specific biological factors, such as the estrogen-mediated regulation of vitamin D receptors, could be involved in the susceptibility to deficiency in chronic HCV [17, 18]. In this research, the mean AST levels (82.4 ± 36.7 IU/L) of patients with severe vitamin D deficiency were much higher than those with no severe deficiency (52.6 ± 23.4 IU/L), indicating a difference of close to 30 IU/L. The same increase was seen in ALT with a value of 74.1 ± 31.5 IU/L vs. 56.3 ± 25.2 IU/L (p=0.014), and was more indicative of active hepatocellular injury. The outcomes are congruent with meta-analytic data that reported negative correlations between serum vitamin D and transaminase activity in chronic viral hepatitis, with pooled correlation coefficients that are between -0.30 and -0.45 [19]. The high levels of total bilirubin also supported the association of liver dysfunction and low vitamin D, whereby the mean of the severe deficiency group was 2.4 ± 1.1mg/dL and the non-severe group was 1.3 ± 0.6mg/dL (p=0.002). consistent with it, Liu *et al.* also documented in a Japanese study that patients who have bilirubin above 2mg/dl are at a higher risk of having severe cases of vitamin D deficiency than patients who have maintained bile flow [20].

This research has some limitations. Firstly, the case-control design cannot be used to make causal inferences, and thus, the results of the observations cannot conclusively prove whether vitamin D deficiency is a cause of disease progression or an effect of more advanced liver dysfunction. The researchers only did the study on one tertiary care center, which could be a problem when it comes to generalizing the results to a larger or more diverse population. A specific Pakistani investigation has never focused on the relationship between cirrhosis and vitamin D deficiency in chronic HCV patients, who are a specific group of subjects under a case-control design. The current regional studies are also restricted to mixed etiologies or not stratified by the severity of deficiencies. Such disparity prevents evidence-based screening and supplementation practices in the local high-burden locations. The present study offers specific information about the association between vitamin D-cirrhosis and the HCV population.

CONCLUSIONS

There was a significant association between vitamin D deficiency and cirrhosis in chronically infected hepatitis C virus patients. Vitamin D deficiency of any severity was observed in 92.0% of the subjects, severe deficiency in almost one-third of cirrhotic patients, versus 14.0% of non-cirrhotic patients. Cirrhosis was the significant determinant of risk of severe vitamin D deficiency more than twice, and a combination of female gender and a longer period of HCV infection added to the risk. Significantly, extreme deficiency of vitamin D was invariably linked to either disturbed biochemical parameter, such as a greater transaminase level, significantly longer international normalized ratio, higher bilirubin, and substantially lower serum albumin, demonstrating advanced hepatocellular damage and synthesizing dysfunction.

Authors' Contribution

Conceptualization: TMC
Methodology: II, JU, JA
Formal analysis: II, JA
Writing and Drafting: HN, SMAR, JA
Review and Editing: HN, TMC, II, JU, SMAR, JA, AUR

All authors approved the final manuscript and take responsibility for the integrity of the work.

Conflicts of Interest

All the authors declare no conflict of interest.

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