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# Understanding Vitamin D Deficiency in Chronic Liver Disease: Exploring the Influence of Demographic, Clinical and Etiological Factors

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**ABSTRACT:** ***Background:*** Vitamin D deficiency is a common issue in patients with chronic liver disease (CLD) and has been linked to various complications. ***Aim:*** This study aimed to assess the prevalence of vitamin D deficiency in CLD patients and explore how demographic, clinical, and etiological factors influence vitamin D status. ***Methods:*** This cross-sectional study was conducted at the Department of Hepatology and Medicine, Rajshahi Medical College Hospital, Bangladesh. A total of 181 patients diagnosed with CLD were selected using purposive sampling. Data collection involved structured interviews, clinical assessments, and laboratory tests, including serum 25-hydroxyvitamin D [25(OH)D] measurement using an enzyme-linked fluorescence assay (ELFA). Statistical analysis was performed using SPSS version 24.0, with Chi-square test used to examine associations between vitamin D levels and various factors. ***Results:*** Vitamin D deficiency was highly prevalent, affecting 81.2% of CLD patients, while 10.5% had insufficient levels, and only 8.3% had sufficient vitamin D levels. Age was significantly linked to vitamin D deficiency ($p = 0.002$), with older patients being more affected, while gender showed no significant association ($p = 0.993$). Disease severity played a crucial role, as patients with ascites ($p = 0.001$) and hepatic encephalopathy ($p = 0.038$) had notably lower vitamin D levels. Among the underlying causes of CLD, hepatitis B virus (HBV) ($p = 0.031$) and non-alcoholic fatty liver disease (NAFLD) ($p = 0.045$) were significantly associated with vitamin D deficiency, whereas hepatitis C virus (HCV) and cryptogenic liver disease also showed high deficiency rates, though without statistical significance. ***Conclusion:*** Older age, severe liver disease complications such as ascites and hepatic encephalopathy, and specific etiologies like HBV and NAFLD are strongly associated with vitamin D deficiency among patients with CLD.

**Keywords:** Vitamin D Deficiency, Chronic Liver Disease, Hepatitis B Virus, Non-Alcoholic Fatty Liver Disease, 25-Hydroxyvitamin D.

**Article at a glance:**
***Study Purpose****: The purpose was to assess Vitamin D deficiency in CLD patients and explore its association with various factors.*
***Key findings:*** *81.2% of patients had Vitamin D deficiency; age, disease severity, and etiology were significant factors.*
***Newer findings:*** *This study reveals that older age, ascites, hepatic encephalopathy, and HBV/NAFLD are associated with Vitamin D deficiency in CLD.*
***Abbreviations:*** *CLD – Chronic Liver Disease, HBV – Hepatitis B Virus, NAFLD – Non-Alcoholic Fatty Liver Disease*

## INRODUCTION

Chronic liver diseases (CLD) remain a major global health concern, ranking among the top causes of death and disability. In 2016, CLD ranked to be 11th leading cause of death (2.2%) and the 15th leading cause of morbidity (1.5% DALYs) worldwide.[1] By 2017, chronic liver disease (CLD) had claimed approximately 1.32 million lives, with men accounting for about two-thirds of these fatalities and women making up the remaining third.[2] More

recently it was estimated that the global burden of chronic liver disease is staggering, affecting an estimated 1.5 billion people across all stages of severity.[3] Among the leading causes, non-alcoholic fatty liver disease (NAFLD) is the most prevalent, responsible for nearly 59% of cases. Hepatitis B virus (HBV) follows, contributing to roughly 29%, while hepatitis C virus (HCV) accounts for about 9% of cases.[2, 4] Other contributing factors include excessive alcohol consumption, autoimmune liver conditions, and metabolic disorders. Viral hepatitis remains the primary driver of liver disease in Bangladesh, with hepatitis B, C, and E being the most common culprits. The prevalence of CLD varies across different regions of the country, ranging between 37% and 69%, with Rajshahi division reporting the highest burden at 69%.[5] Vitamin D deficiency is extremely common among individuals with chronic liver disease (CLD), regardless of the underlying cause. Research suggests that up to 93% of CLD patients experience some degree of vitamin D insufficiency.[6, 7] Impaired liver function affects the hydroxylation Vitamin D deficiency is extremely common among individuals with chronic liver disease (CLD), regardless of the underlying cause. Research suggests that up to 93% of CLD patients experience some degree of vitamin D insufficiency.[6, 7] Impaired liver function affects the hydroxylation process necessary for converting vitamin D into its active form, while also reducing the production of albumin and vitamin D-binding protein (DBP), both of which are important for transporting and maintaining vitamin D in circulation.[8, 9]

However, vitamin D deficiency in CLD is not solely due to impaired liver synthesis. It is also highly prevalent among patients without cirrhosis, suggesting additional contributing factors such as poor dietary intake, malabsorption, and decreased sun exposure. Hypovitaminosis Vit D among CLD patients is not merely a consequence of impaired liver function but is also significantly influenced by demographic, clinical, and etiological factors.[10] Insufficient vitamin D levels in CLD have been linked to an increased risk of bacterial infections and complications associated with portal hypertension.[11, 12] These levels decline even further in advanced cirrhosis as hepatic dysfunction worsens.[13, 14] Therefore, vitamin D deficiency in CLD is driven by multiple factors beyond hepatic impairment alone. Demographic variables such as age, gender, and geographical location; clinical factors including obesity, diabetes, and malabsorption issues; and variations in the underlying causes of liver disease, such as viral hepatitis, NAFLD, or alcohol-related liver disease, all play a significant role in determining vitamin D status.[15, 16] Recognizing these contributing factors is essential for understanding the complexity of Vit D deficiency in CLD patients and for developing targeted prevention and management strategies. These variations hare unexplored territory in Bangladesh. Therefore, the present study aimed to assess the prevalence of Vit D deficiency in patients with CLD and explore how demographic, clinical, and etiological factors influence vitamin D status.

**Aims And Objectives**

To assess the prevalence of Vit D deficiency in CLD patients and explore how demographic, clinical, and etiological factors influence vitamin D status.

## MATERIALS AND METHODS

This cross-sectional descriptive study was conducted at the Department of Hepatology and Medicine, Rajshahi Medical College Hospital, Rajshahi, Bangladesh. Before commencing the study, ethical approval was obtained from the Ethics and Research Committee of Rajshahi Medical College Hospital. Written informed consent was taken from all participants or their responsible family members after explaining the study's purpose, procedures, and potential risks. Participants were assured that their participation was voluntary, and they had the right to withdraw at any time without affecting their medical care. Confidentiality of patient data was strictly maintained, and no financial incentives were provided to participants. The study population consisted of patients diagnosed with chronic liver disease (CLD) who were admitted to the Medicine and Hepatology department. A purposive sampling technique was used to select the participants, and according to calculated sample size, a total of 181 CLD patients were selected for the study. Patients aged 18 years or older with a confirmed diagnosis of CLD were included in the study. However, those receiving medications that could influence serum vitamin D levels, such as calcium and vitamin D supplements, steroids, antiepileptic drugs, and bisphosphonates, were excluded. Additionally, patients with chronic conditions affecting calcium and vitamin D metabolism, including chronic kidney disease, malabsorption syndrome, and tuberculosis, as well as those diagnosed with hepatocellular carcinoma or

other malignancies, and pregnant women, were also excluded.

**Data Collection**

Data collection was conducted through structured interviews, clinical examinations, and laboratory investigations. A pretested questionnaire was used to gather socio-demographic data, clinical history, and biochemical parameters. The clinical diagnosis of cirrhosis was based on a combination of clinical, biochemical, and presence of ascites, splenomegaly and hepatic encephalopathy. Serum 25-hydroxyvitamin D [25(OH)D] levels were measured using the VIDAS system, which employs an enzyme-linked fluorescence assay (ELFA). All collected data were entered, tabulated, and verified for accuracy before being analyzed using SPSS version 24.0. Quantitative data were expressed as mean and standard deviation, while categorical variables were presented as frequencies and percentages. The Chi-square ($\chi^2$) test was used for categorical comparisons, and p-value of <0.05 was shown as statistically significant finding.

## RESULTS

A majority of the study participants (81.2%) were vitamin D deficient, while 10.5% had insufficient levels and only 8.3% had sufficient levels (figure 1).

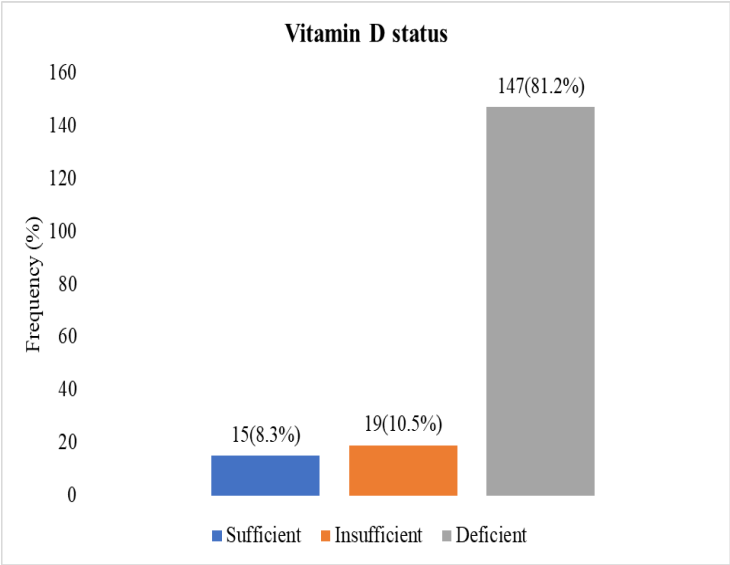


**Figure 1: Distribution of the Participants According to Vitamin D Deficiency in Chronic Liver Disease (N=181)**

**Influence of Demographic Factors on Vitamin D Deficiency**

Age showed a significant association with vitamin D deficiency (p = 0.002), with older individuals being more affected. The highest deficiency rates were observed in patients aged 61-70 years (91.3%) and those above 70 years (83.3%). Sex, however, was not significantly associated with vitamin D deficiency (p = 0.993), as both males (81.3%) and females (81%) exhibited similar deficiency rates (table 1).

**Table 1: Distribution of the Participants According to Influence of Demographic Factors on Vit D Deficiency Among Patients with CLD (N=181)**

| Demographic factors | Vit D level | | | P value |
|---|---|---|---|---|
| | Sufficient | Insufficient | Deficient | |
| **Age** | | | | |
| 18-30 | 5 (38.5%) | 2 (15.4%) | 6 (46.2%) | |
| 31-40 | 5 (13.2%) | 2 (5.3%) | 31 (81.6%) | |
| 41-50 | 0 | 4 (12.1%) | 29 (87.9%) | **0.002[a]** |
| 51-60 | 3 (4.8%) | 9 (14.5%) | 50 (80.6%) | |

| | | | | |
|---|---|---|---|---|
| 61-70 | 2 (8.7%) | 0 | 21 (91.3%) | |
| >70 | 0 | 2 (16.7%) | 10 (83.3%) | |
| **Sex** | | | | |
| Female | 5 (8.6%) | 6 (10.3%) | 47 (81%) | 0.993[a] |
| Male | 10 (8.1%) | 13 (10.6%) | 100 (81.3%) | |

a= chi square test

**Influence of Clinical Factors on Vitamin D Deficiency**

The severity of ascites had a significant impact on vitamin D levels (p = 0.001), with higher deficiency rates in patients with mild (88%) and marked ascites (88.3%) compared to those without ascites (55.3%). Hepatic encephalopathy was also significantly associated with vitamin D deficiency (p = 0.038), with 92.6% of affected patients being deficient compared to 76.4% of those without encephalopathy (Table 2).

**Table 2: Distribution of the Participants According to Influence of Clinical Factors on Vit D Deficiency Among Patients with CLD (N=181)**

| Clinical factors | Vit D level | | | P value |
|---|---|---|---|---|
| | Sufficient | Insufficient | Deficient | |
| **Ascites grade** | | | | |
| None | 12 (31.6%) | 5 (13.2%) | 21 (55.3%) | |
| Mild | 3 (3.6%) | 7 (8.4%) | 73 (88%) | **0.001**[a] |
| Marked | 0 | 7 (11.7%) | 53 (88.3%) | |
| **Hepatic encephalopathy** | | | | |
| Yes | 2 (3.7%) | 2 (3.7%) | 50 (92.6%) | **0.038**[a] |
| No | 13 (10.2%) | 17 (13.4%) | 97 (76.4%) | |

a= chi square test

**Influence of Etiological Factors on Vitamin D Deficiency**

Patients with hepatitis B virus (HBV) infection had a significantly high prevalence of vitamin D deficiency (75.6%, p = 0.031). Non-alcoholic fatty liver disease (NAFLD) was also significantly associated with deficiency (84.5%, p = 0.045). Although 91.4% of hepatitis C virus (HCV) patients were deficient, the association was not statistically significant (p = 0.077). Similarly, vitamin D deficiency was observed in 100% of cryptogenic CLD cases and 71.4% of Wilson's disease cases, but these findings were not statistically significant (p = 0.074 and p = 0.107, respectively) (Table 3).

**Table 3: Distribution of the Participants According to Influence of Etiological Factors on Vit D Deficiency Among Patients with CLD (N=181)**

| Etiological factors | Vit D level | | | P value |
|---|---|---|---|---|
| | Sufficient | Insufficient | Deficient | |
| HBV | 6 (7.3%) | 14 (17.1%) | 62 (75.6%) | **0.031**[a] |
| HCV | 3 (8.6%) | 0 | 32 (91.4%) | 0.077[a] |
| NAFLD | 4 (3.9%) | 12 (11.7%) | 87 (84.5%) | **0.045**[a] |
| Wilson disease | 2 (28.6%) | 0 | 5 (71.4%) | 0.107[a] |
| Cryptogenic | 0 | 0 | 20 (100%) | 0.074[a] |

*a= chi square test*

## DISCUSSION

The present study highlighted the high prevalence of vitamin D deficiency among patients with chronic liver disease (CLD) and its association with various demographic, clinical, and etiological factors. A significant proportion of participants

(81.2%) were vitamin D deficient, with only 8.3% having sufficient levels. This finding is consistent with previous studies that have reported a high prevalence of vitamin D deficiency in CLD, reinforcing the notion that liver dysfunction significantly impacts vitamin D metabolism.[14, 17–22] Age was found to be a significant factor influencing vitamin D levels, with older individuals exhibiting higher rates of deficiency. The prevalence was highest among patients aged 61-70 years (91.3%) and those above 70 years (83.3%) ($p = 0.002$). This was in line with existing research that associates aging with reduced cutaneous synthesis of vitamin D and decreased hepatic hydroxylation, further exacerbating the deficiency in CLD patients.[8, 14, 16, 22–26] However, no significant difference in vitamin D levels was observed between males and females ($p = 0.993$), suggesting that gender does not play a crucial role in determining vitamin D status among CLD patients. But male prevalence was found in patients with CLD. In general, men are 2 fold more likely to die from CLD and cirrhosis of liver than are women, according to an analysis by the National Center for Health Statistics that was reported in 2005.[27, 28]

The clinical severity of liver disease seemed to have a significant impact on vitamin D levels. Ascites, a key marker of liver dysfunction, showed a strong correlation with vitamin D deficiency ($p = 0.001$). Patients with mild (88%) and marked ascites (88.3%) exhibited significantly lower vitamin D levels compared to those without ascites (55.3%). Similarly, hepatic encephalopathy was associated with severe vitamin D deficiency ($p = 0.038$), with 92.6% of affected patients having deficient levels. These findings suggest that worsening liver function leads to impaired vitamin D metabolism, possibly due to reduced hepatic conversion of vitamin D into its active form and increased sequestration in ascitic fluid. Etiological factors also played a role in vitamin D deficiency among CLD patients. Hepatitis B virus (HBV) infection was significantly associated with vitamin D deficiency ($p = 0.031$), with 75.6% of HBV patients exhibiting deficient levels. Several other literatures showed similar observation that Vit D deficiency is common in HBV infection and CLD.[29, 30] Non-alcoholic fatty liver disease (NAFLD) was significantly correlated with vitamin D deficiency ($p = 0.045$), with 84.5% of NAFLD patients having low vitamin D levels. This indicated towards the hypothesis that low vit D level coexists with NAFLD and nano emulsion of Vit D have a hepatoprotective effect.[31] Similar observations were also seen by numerous cohort studies.[32–34] Although hepatitis C virus (HCV) infection showed a high prevalence of deficiency (91.4%), the association was not statistically significant ($p = 0.077$). Additionally, 100% of patients with cryptogenic CLD and 71.4% of those with Wilson's disease were vitamin D deficient, though these findings did not reach statistical significance. The high prevalence of deficiency among these subgroups indicates that underlying liver pathology influences vitamin D metabolism, possibly due to chronic inflammation, impaired bile acid production, and hepatic fibrosis. On the contrary, evidence was present where HCV was found to be prevalent etiology.[35] Overall, this study underscores the need for routine vitamin D screening and supplementation in CLD patients, particularly those with advanced disease, ascites, hepatic encephalopathy, or HBV and NAFLD as underlying etiologies. Addressing vitamin D deficiency in CLD patients may have potential therapeutic implications, including improved bone health, reduced risk of hepatic osteodystrophy, and better overall disease prognosis.

## CONCLUSION

This explorative study observed that older age, severe liver disease complications such as ascites and hepatic encephalopathy, and specific etiologies like HBV and NAFLD are strongly associated with vitamin D deficiency among patients with CLD.

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