International Journal of Medical and Pharmaceutical Research

Online ISSN-2958-3683 | Print ISSN-2958-3675
Frequency: Bi-Monthly
Available online on: https://ijmpr.in/

Research Article

STUDY OF VITAMIN D3 AND VITAMIN B12 LEVELS IN PATIENTS WITH CHRONIC LIVER DISEASE

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Received: 13-09-2025
Accepted: 26-09-2025
Available online: 13-10-2025

ABSTRACT

Background: Chronic liver disease (CLD) is a major global health problem characterized by progressive hepatic dysfunction affecting vitamin metabolism. The liver plays a crucial role in the hydroxylation of vitamin D3 and storage of vitamin B12; hence, their deficiencies are common in CLD and may influence disease progression.
Objectives: To evaluate serum vitamin D3 and vitamin B12 levels in patients with CLD and to determine their association with disease severity and biochemical parameters.
Methods: An observational longitudinal study was conducted on 120 CLD patients aged 18–65 years in the Department of Biochemistry, MGM Medical College & LSK Hospital, Kishanganj, Bihar. Serum vitamin D3 (25-OH-D) and vitamin B12 levels were measured using CLIA and ECLIA methods, respectively. Liver function tests and disease severity (Child-Pugh and MELD scores) were assessed. Statistical analysis was performed using SPSS v26.0; $p < 0.05$ was considered significant.
Results: Most patients were middle-aged (31–60 years: 75%) and male (70%). Hepatitis B/C (45%) and alcoholic liver disease (30%) were the predominant etiologies. Vitamin D3 deficiency (<20 ng/mL) was observed in 60% and insufficiency in 25% of patients. Vitamin B12 levels were normal in 40%, low in 35%, and elevated in 25%. Vitamin D3 showed significant negative correlations with AST ($r = –0.48$) and INR ($r = –0.55$) and a positive correlation with albumin ($r = +0.61$). Vitamin B12 correlated weakly but significantly with AST and INR.
Conclusion: Vitamin D3 deficiency and altered vitamin B12 levels are highly prevalent in CLD and closely associated with disease severity. Regular monitoring and correction of these micronutrients may help improve clinical outcomes in chronic liver disease patients.

Keywords: *Chronic Liver Disease (CLD); Vitamin D3; 25-Hydroxyvitamin D; Vitamin B12; Cobalamin; Child-Pugh Score; MELD Score; Liver Function Tests.*

INTRODUCTION

Chronic liver disease (CLD) represents a significant global health burden, encompassing a spectrum of conditions such as hepatitis, cirrhosis, and hepatocellular carcinoma, which progressively impair liver function.[1] The liver plays a central role in metabolic homeostasis, including the synthesis, storage, and activation of essential vitamins.[2] Among these, vitamin D3 (cholecalciferol) and vitamin B12 (cobalamin) are critical micronutrients whose metabolism is heavily dependent on hepatic integrity.[3] Deficiencies in these vitamins have been increasingly recognized as common complications in CLD, contributing to disease progression and associated morbidity.[4] Given the liver's pivotal role in vitamin D hydroxylation and vitamin B12 storage, impaired hepatic function may lead to significant disruptions in their bioavailability, warranting further investigation into their levels in CLD patients.[5]

Vitamin D3, a fat-soluble vitamin, undergoes hydroxylation in the liver to form 25-hydroxyvitamin D [25(OH)D], the major circulating and biologically active metabolite.[6] This conversion is crucial for maintaining calcium homeostasis, bone health, and immune modulation.[7] However, in CLD, reduced hepatic function can diminish the synthesis of 25(OH)D, leading to vitamin D deficiency, which has been linked to increased risks of osteopenia, muscle weakness, and enhanced inflammatory responses.[8] Epidemiological studies have reported a high prevalence of vitamin D deficiency in CLD patients, with severity often correlating with the degree of liver dysfunction.[9] Furthermore, emerging evidence suggests that vitamin D may exert hepatoprotective effects by modulating fibrogenic pathways, thereby influencing disease progression.[10] Despite these findings, the precise mechanisms underlying vitamin D deficiency in CLD and its clinical implications remain incompletely understood, necessitating further research.[11]

Similarly, vitamin B12, a water-soluble vitamin, is stored primarily in the liver and is essential for DNA synthesis, red blood cell formation, and neurological function.[12] The liver's role in B12 metabolism involves the uptake and storage of cobalamin, which is released into circulation as needed.[13] In CLD, impaired hepatic storage capacity and altered release mechanisms may lead to either deficiency or, paradoxically, elevated serum levels due to reduced tissue utilization and hepatocellular damage.[14] Studies have reported mixed findings regarding vitamin B12 status in CLD, with some indicating deficiency due to malabsorption and others showing elevated levels resulting from hepatic necrosis and subsequent release of stored B12.[15] This discrepancy highlights the complexity of vitamin B12 metabolism in liver disease and underscores the need for a more comprehensive assessment of its levels in different stages of CLD.[15]

The interplay between vitamin D3 and vitamin B12 deficiencies in CLD may also have synergistic detrimental effects on patient outcomes. For instance, vitamin D deficiency has been associated with increased fatigue and musculoskeletal symptoms, while B12 deficiency can exacerbate anemia and neurological complications, collectively diminishing the quality of life in CLD patients. Additionally, both vitamins play roles in modulating oxidative stress and inflammation, which are key drivers of liver fibrosis and cirrhosis. Therefore, evaluating their levels could provide valuable insights into disease severity and potential therapeutic interventions by elucidating the prevalence and impact of these deficiencies, this research seeks to contribute to a better understanding of their role in CLD progression and inform potential therapeutic strategies. The findings may also pave the way for standardized screening protocols and personalized supplementation approaches to improve patient management.

Objectives:
To evaluate serum vitamin D3 and vitamin B12 levels in patients with CLD and to determine their association with disease severity and biochemical parameters.

MATERIALS & METHODS

Study Design:

- **Type of Study**: Observational longitudinal study.
- **Duration**: The study was conducted over a period of **22 months (July** 2023 to April 2025) to including patient recruitment, follow-up, and data analysis.
- **Setting**: The study was conducted in the **Department of Biochemistry, MGM Medical College & LSK Hospital, Kishanganj, Bihar**, in collaboration with the Department of Medicine.

Study Population

- **Inclusion Criteria**:
  - Patients aged 18–65 years diagnosed with chronic liver disease (CLD) based on clinical, biochemical, and imaging findings.
  - Etiologies of CLD include viral hepatitis (HBV, HCV), alcoholic liver disease, non-alcoholic fatty liver disease (NAFLD), or cirrhosis of any cause.
  - Willing to provide informed consent for participation in the study.
- **Exclusion Criteria**:
  - Patients with acute liver disease or acute-on-chronic liver failure.
  - Patients with other systemic diseases (e.g., chronic kidney disease, malignancy, or hematological disorders).
  - Patients on vitamin B12 supplementation or multivitamin therapy in the past 6 months.
  - Pregnant or lactating women.

Sample Size
Overall prevalence of CLD in Bihar reported by Pandey et al in 2021. i.e. 24.8% according to formula:

$$n = \frac{[Z\ (1-\alpha/2)]^2 \cdot P \cdot (1-P)}{(\varepsilon)^2}$$

$[Z\ (1-\alpha/2)] = 1.96$
$P = 24.8\% = (i.e.\ 0.248)$
$1-P = 0.752$

Relative precision (ε) = 8% (i.e.(0.08)
Calculated sample size 111.9 and The final rounded sample size is 1**20.**

Data Collection & Biochemical Analysis
Baseline Assessment
Demographic & Clinical Data:
- Age, gender, etiology of CLD, duration of disease, alcohol/tobacco history.
- **Child-Pugh score, MELD score**, and clinical symptoms (jaundice, ascites, hepatic encephalopathy).

Blood Sampling & Laboratory Tests:
- 5 mL venous blood collected under aseptic conditions.
- Serum separation by centrifugation (3000 rpm, 10 min) and stored at -80°C until analysis.

Biochemical Parameters:
- Liver Function Tests (LFTs): Bilirubin, AST, ALT, ALP, albumin, PT, INR.
- Vitamin D3 (25-OH-D): Measured by chemiluminescence immunoassay (CLIA) (normal: 30-100 ng/mL; deficiency: <20 ng/mL).
- Vitamin B12 (cobalamin): Measured by electrochemiluminescence immunoassay (ECLIA) (normal: 200-900 pg/mL; deficiency: <200 pg/mL).

Follow-Up Assessments
- Patients were followed up every 3 months for 12 months to track:
- Changes in vitamin D3/B12 levels.
- Disease progression (Child-Pugh/MELD score changes).

Statistical Analysis
Data analyzed using SPSS v26.0 and GraphPad Prism. Descriptive statistics (mean ± SD, percentages). Comparative analysis was done Student's t-test/Mann-Whitney U test (for continuous variables). And Chi-square test (for categorical variables). Pearson/Spearman test for vitamin levels vs. disease severity. P value <0.05 was consider as statiticall significant.

Ethical Considerations
Approved by the Institutional Ethics Committee (IEC). Written informed consent obtained from all participants. Confidentiality maintained as per Helsinki Declaration.

RESULTS & ANALYSIS:

Table 1: Age Distribution of CLD Patients (n=120)

Age Group (Years)	Number of Patients (n)	Percentage (%)
18–30	18	15.0
31–45	42	35.0
46–60	48	40.0
>60	12	10.0

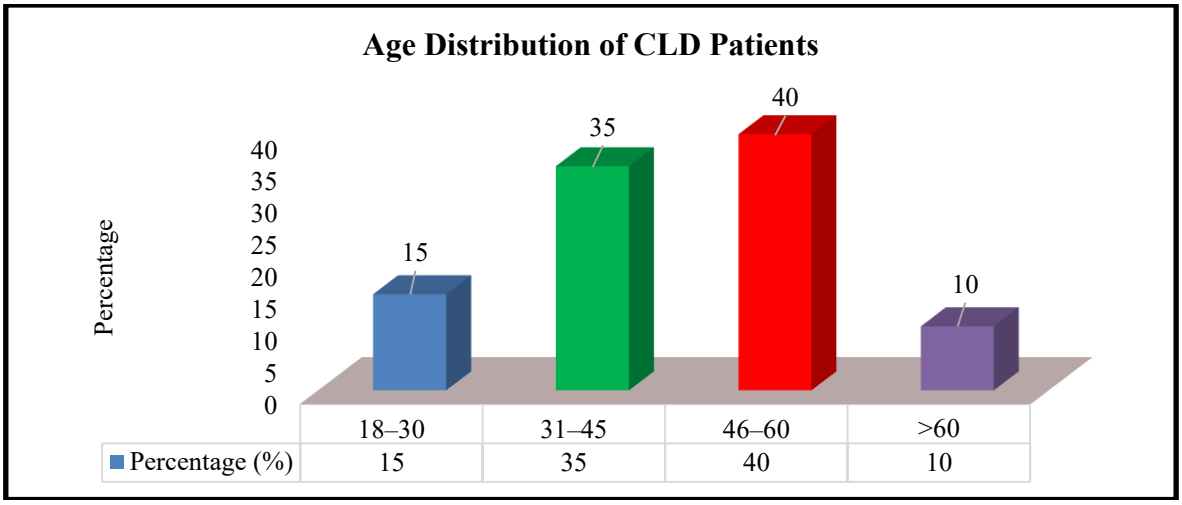

Figure 1: Age Distribution of CLD Patients (n=120)

In the present study comprising 120 patients with chronic liver disease, the age distribution revealed that 15.0% of patients were in the 18–30 years age group, 35.0% were between 31–45 years, and 40.0% belonged to the 46–60 years age group. Patients aged above 60 years constituted the remaining 10.0% of the study population.

Table 2: Sex Distribution of CLD Patients (n=120)

Sex	Number of Patients (n)	Percentage (%)
Male	84	70.0
Female	36	30.0

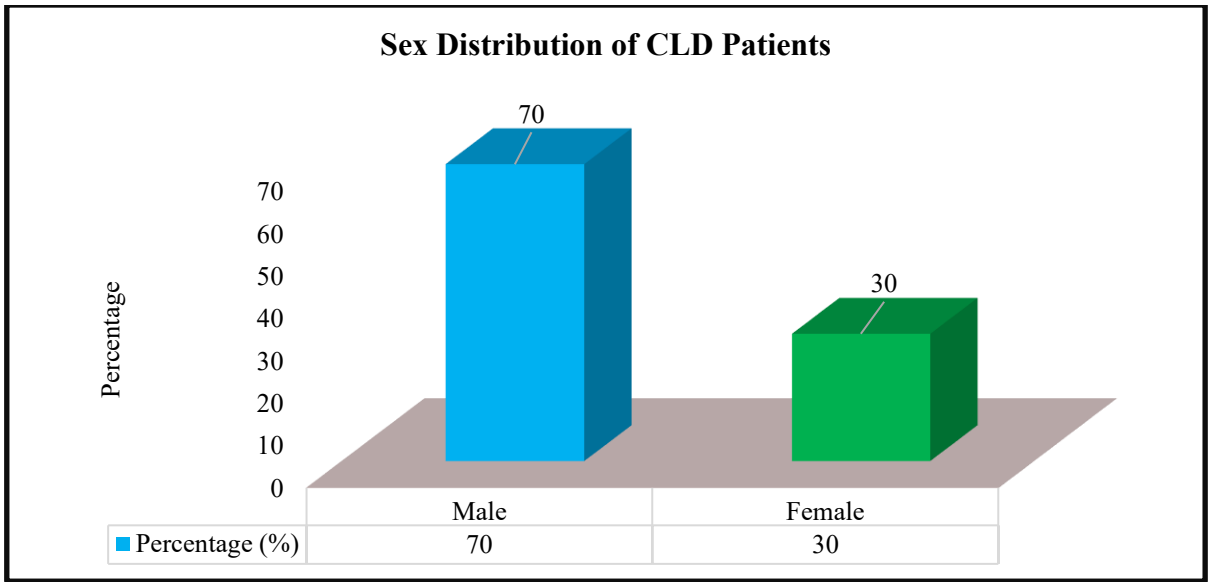

Figure 2: Sex Distribution of CLD Patients (n=120)

Among the 120 patients with chronic liver disease in the study, 70.0% were males (84 patients) and 30.0% were females (36 patients),

Table 3: Underlying Causes of CLD (n=120)

Etiology	Number of Patients (n)	Percentage (%)
Hepatitis B/C	54	45.0
Alcoholic Liver Disease	36	30.0
NAFLD/NASH	18	15.0
Cryptogenic Cirrhosis	12	10.0

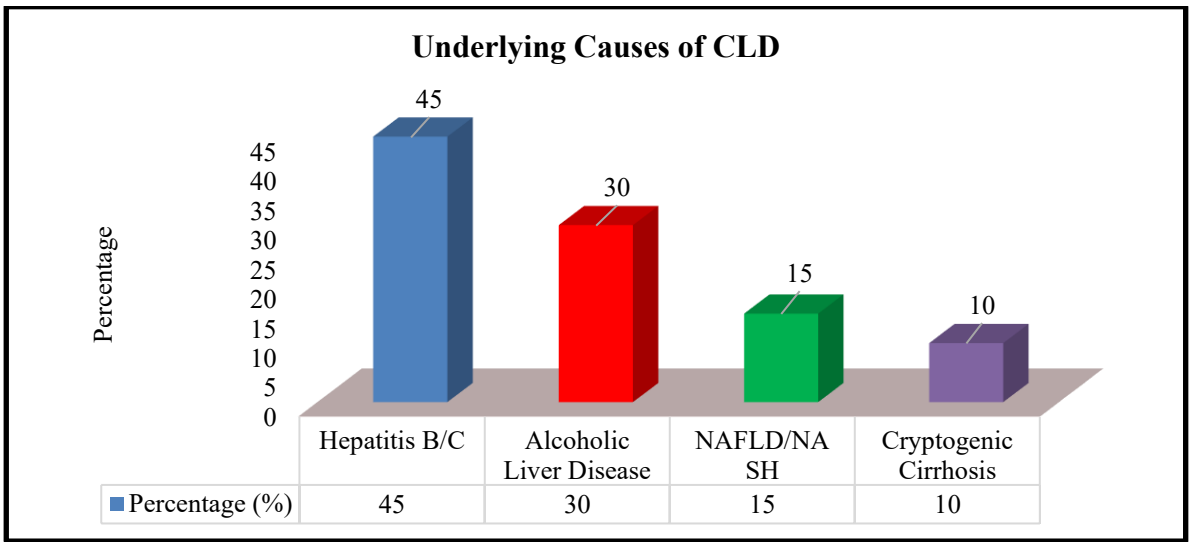

Figure 3: Underlying Causes of CLD (n=120)

Regarding the underlying causes of chronic liver disease among the study participants, Hepatitis B or C infection was the most common etiology, accounting for 45.0% (54 patients). Alcoholic liver disease was the second most frequent cause, observed in 30.0% (36 patients). Non-alcoholic fatty liver disease (NAFLD) or non-alcoholic steatohepatitis (NASH) contributed to 15.0% (18 patients), while cryptogenic cirrhosis was identified in 10.0% (12 patients) of the cases.

Table 4: Child-Pugh Classification (n=120)

Child-Pugh Class	Number of Patients (n)	Percentage (%)
Class A (Mild)	36	30.0
Class B (Moderate)	60	50.0
Class C (Severe)	24	20.0

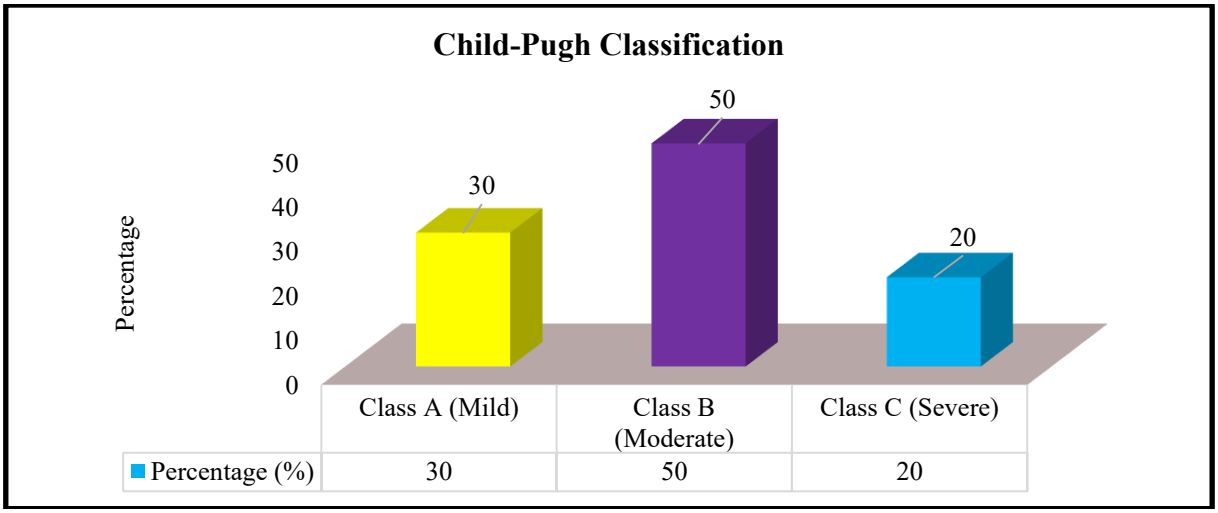

Figure 4: Child-Pugh Classification (n=120)

Assessment of disease severity using the Child-Pugh classification showed that 30.0% (36 patients) were in Class A indicating mild disease, 50.0% (60 patients) were categorized as Class B representing moderate disease, while 20.0% (24 patients) were in Class C signifying severe disease.

Table 5: MELD Score Distribution (n=120)

MELD Score Range	Number of Patients (n)	Percentage (%)
≤15 (Low risk)	30	25.0
16–25 (Moderate)	66	55.0
≥26 (High risk)	24	20.0

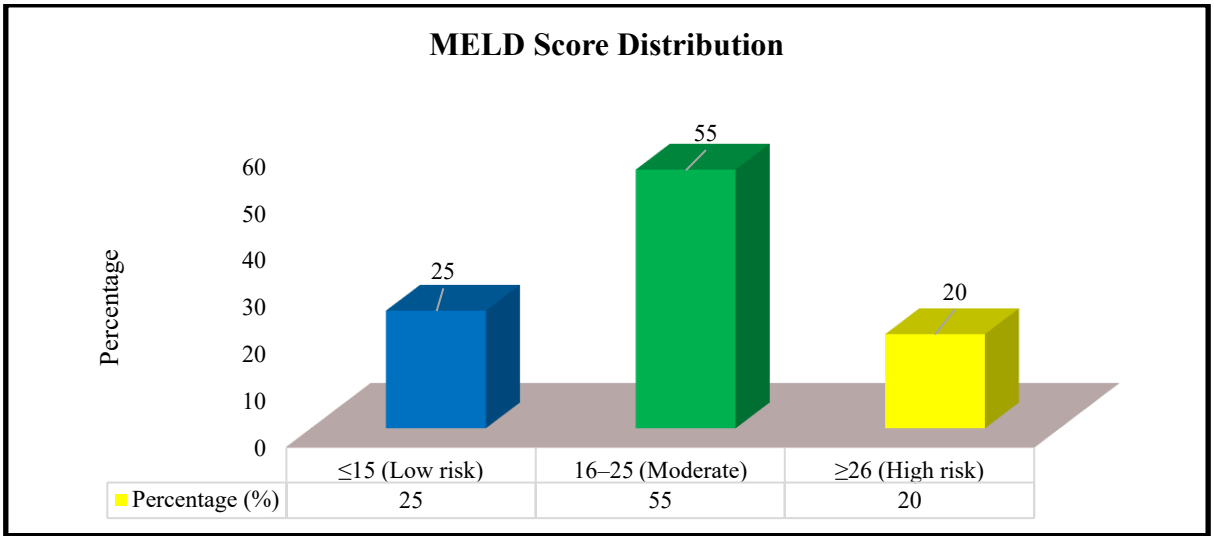

Figure5: MELD Score Distribution (n=120)

Based on the MELD score assessment, 25.0% of patients (30 individuals) had a score of 15 or less, indicating low risk. A majority of 55.0% (66 patients) had MELD scores ranging between 16 and 25, corresponding to moderate risk, while 20.0% (24 patients) had scores of 26 or higher.

Table 6: Vitamin D3 Levels in CLD Patients (n=120)

Vitamin D3 Status	Number of Patients (n)	Percentage (%)

Normal (≥30 ng/mL)	18	15.0
Insufficient (20–29 ng/mL)	30	25.0
Deficient (<20 ng/mL)	72	60.0

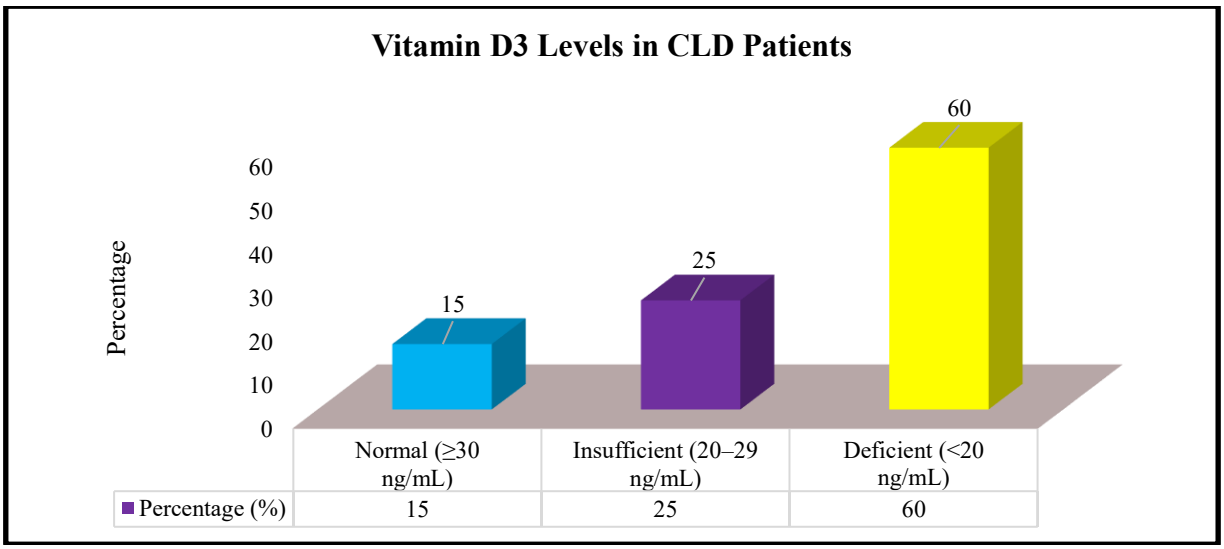

Figure 6: Vitamin D3 Levels in CLD Patients (n=120)

Evaluation of Vitamin D3 levels among the patients revealed that only 15.0% (18 patients) had normal levels (≥30 ng/mL), whereas 25.0% (30 patients) were found to have insufficient levels (20–29 ng/mL). A significant majority, 60.0% (72 patients), were deficient in Vitamin D3 with levels below 20 ng/mL.

Table 7: Vitamin B12 Levels in CLD Patients (n=120)

Vitamin B12 Status	Number of Patients (n)	Percentage (%)
Normal (200–900 pg/mL)	48	40.0
Low (<200 pg/mL)	42	35.0
High (>900 pg/mL)	30	25.0

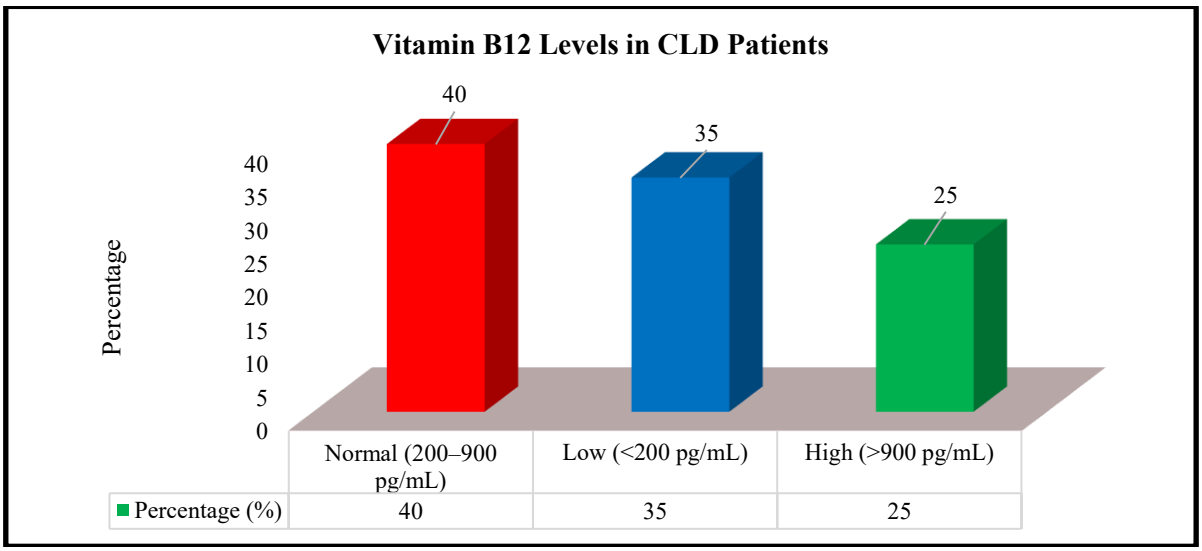

Figure 7: Vitamin B12 Levels in CLD Patients (n=120)

Analysis of Vitamin B12 levels showed that 40.0% of patients (48 individuals) had values within the normal range (200–900 pg/mL), while 35.0% (42 patients) exhibited low Vitamin B12 levels (<200 pg/mL). Additionally, 25.0% (30 patients) had elevated Vitamin B12 levels (>900 pg/mL).

Table 8: Vitamin D3/B12 vs. Child-Pugh Class

Parameter	Child-Pugh A (n=36)	Child-Pugh B (n=60)	Child-Pugh C (n=24)	p-value
Vitamin D3 (ng/mL)	28.5 ± 6.2	18.4 ± 5.8	12.1 ± 4.3	**<0.001**

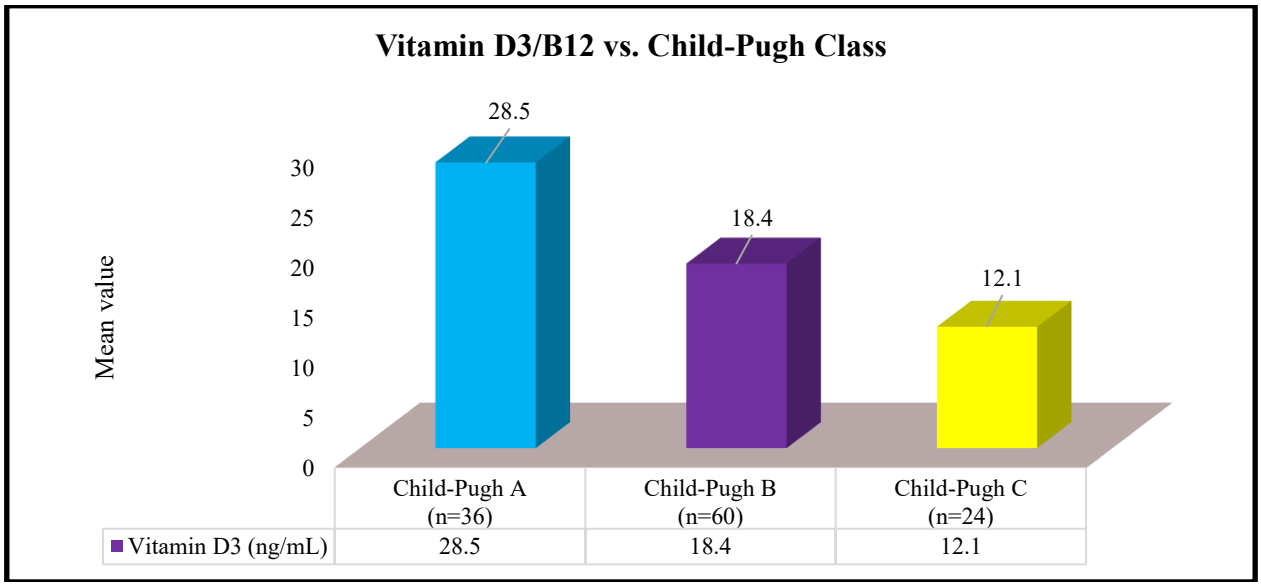

Figure 8: Vitamin D3/B12 vs. Child-Pugh Class

The mean Vitamin D3 levels were found to decrease progressively with worsening Child-Pugh class. Patients in Child-Pugh Class A had a mean Vitamin D3 level of 28.5 ± 6.2 ng/mL, which declined to 18.4 ± 5.8 ng/mL in Class B and further to 12.1 ± 4.3 ng/mL in Class C. This difference across the groups was statistically significant, with a p-value of <0.001.

Table 9: Mean LFT Parameters Across Disease Severity

Parameter	Normal Range	All CLD Patients (n=120)	Child-Pugh A (n=36)	Child-Pugh B (n=60)	Child-Pugh C (n=24)	p-value
Total Bilirubin (mg/dL)	0.2–1.2	1.3 ± 1.9	1.3 ± 0.6	1.39 ± 1.2	1.48 ± 2.1	**0.001**
Direct Bilirubin (mg/dL)	0–0.3	0.80 ± 1.2	0.8 ± 0.3	0.87 ± 0.8	0.92 ± 1.5	0.068
AST (U/L)	10–40	128 .2± 62.7	68.2 ± 28.3	99.6 ± 45.7	122.4 ± 75.1	**<0.001**
ALT (U/L)	7–56	85.3 ± 42.1	52 ± 20	88 ± 30	140 ± 58	**<0.001**
ALP (U/L)	44–147	240 ± 110	160 ± 65	174.2 ± 90	195.2 ± 84.2	**<0.001**
Albumin (g/dL)	3.5–5.0	2.8 ± 0.7	3.5 ± 0.4	2.7 ± 0.5	2.3 ± 0.3	**<0.001**
INR	0.8–1.2	1.8 ± 0.5	1.2 ± 0.2	1.8 ± 0.3	2.5 ± 0.6	**<0.001**

In the overall study population of chronic liver disease patients, the mean total bilirubin level was slightly elevated at 1.3 ± 1.9 mg/dL compared to the normal range (0.2–1.2 mg/dL), with a progressive increase observed from Child-Pugh A (1.3 ± 0.6 mg/dL) to Child-Pugh C (1.48 ± 2.1 mg/dL), showing a statistically significant difference (p = 0.001).Direct bilirubin levels were also raised overall (0.80 ± 1.2 mg/dL), but the difference among the Child-Pugh classes was not statistically significant (p = 0.068).AST levels were markedly elevated (128.2 ± 62.7 U/L), rising from 68.2 ± 28.3 U/L in Class A to 122.4 ± 75.1 U/L in Class C, with a highly significant p-value (<0.001).Similarly, ALT levels showed a progressive increase from 52 ± 20 U/L in Class A to 140 ± 58 U/L in Class C (p < 0.001).Alkaline phosphatase (ALP) levels were elevated across all groups, with a mean value of 240 ± 110 U/L overall, again showing a significant upward trend with worsening Child-Pugh class (p < 0.001).Serum albumin levels decreased progressively from 3.5 ± 0.4 g/dL in Class A to 2.3 ± 0.3 g/dL in Class C (p < 0.001), indicating deteriorating synthetic liver function with disease severity. Finally, the mean INR was raised (1.8 ± 0.5), increasing significantly from 1.2 ± 0.2 in Class A to 2.5 ± 0.6 in Class C, reflecting worsening coagulopathy with disease progression (p < 0.001).

Table 10: Correlation of LFTs with Vitamin D3/B12 Levels

LFT Parameter	Vitamin D3 (r-value)	p-value	Vitamin B12 (r-value)	p-value
Total Bilirubin	-0.52	**0.097**	0.18	0.12
AST	-0.48	**<0.001**	0.22*	**0.03**
Albumin	+0.61	**<0.001**	-0.15	0.18
INR	-0.55	**<0.001**	0.25*	**0.02**

Correlation analysis revealed that Vitamin D3 levels had a significant negative correlation with AST ($r = –0.48$, $p < 0.001$) and INR ($r = –0.55$, $p < 0.001$), while showing a significant positive correlation with albumin levels ($r = +0.61$, $p < 0.001$). Although a negative correlation was observed between Vitamin D3 and total bilirubin ($r = –0.52$), it did not reach statistical significance ($p = 0.097$). Regarding Vitamin B12 levels, a weak but significant positive correlation was found with AST ($r = +0.22$, $p = 0.03$) and INR ($r = +0.25$, $p = 0.02$). No statistically significant correlation was observed between Vitamin B12 and total bilirubin ($r = +0.18$, $p = 0.12$) or albumin ($r = –0.15$, $p = 0.18$).

DISCUSSION

In this study of 120 chronic liver disease (CLD) patients, most were middle-aged (31–60 years: 75%), with male predominance (70%), consistent with findings by Aziz et al.[16] and Islam Shah et al.[17] The common etiologies were Hepatitis B/C (45%), alcoholic liver disease (30%), and NAFLD (15%), similar to Islam Shah et al.[17]

Based on Child-Pugh classification, 30% were Class A, 50% Class B, and 20% Class C, comparable to Scheiner et al. [18]. MELD scoring showed 55% of patients in the moderate-risk range (16–25).

Vitamin D3 deficiency (<20 ng/mL) was seen in 60% of patients, aligning with Abbasi et al. [19], Khan et al. [20], Lower Vitamin D levels correlated negatively with Child-Pugh and MELD scores, confirming earlier findings by Ravaioli et al. [21] and Adiri et al. [22].

Vitamin B12 levels were normal in 40%, low in 35%, and elevated in 25%—similar to Kumar et al. [23], who attributed hypercobalaminemia to hepatic leakage. Dastidar et al. [24] also reported comparable B12 deficiency rates.

Biochemically, AST, ALT, ALP, bilirubin, and INR levels increased with disease severity, while albumin declined significantly ($p < 0.001$), consistent with Aziz et al. [16], Scheiner et al. [18], and Gad et al. [25].

Vitamin D3 showed strong negative correlations with AST ($r = –0.48$) and INR ($r = –0.55$) and a positive correlation with albumin ($r = +0.61$), reaffirming associations reported by Khan et al. [26] and Adiri et al. [22]. Vitamin B12 correlated weakly but positively with AST and INR, supporting Kumar et al. [23].

Overall, declining Vitamin D3 and abnormal Vitamin B12 profiles were closely linked with advancing liver dysfunction, underscoring their potential role as biochemical markers in chronic liver disease.

CONCLUSION

In this study involving 120 patients with chronic liver disease, the majority of patients were middle-aged adults (31–60 years), with a clear male predominance. Hepatitis B and C infections emerged as the most common etiological factors, followed by alcoholic liver disease and non-alcoholic fatty liver disease.

The severity of liver dysfunction, assessed through Child-Pugh and MELD scores, indicated that most patients had moderate disease, although a significant proportion had progressed to severe stages.

A striking finding was the high prevalence of Vitamin D3 deficiency, observed in 60% of the study population, with Vitamin D3 levels showing a significant decline as the severity of liver disease increased. Similarly, Vitamin B12 abnormalities were frequent, with 35% of patients demonstrating deficiency and 25% showing elevated levels. Liver function parameters worsened progressively with advancing Child-Pugh class, with significant increases in bilirubin, AST, ALT, ALP, and INR levels, along with declining serum albumin. Correlation analysis highlighted a significant negative association between Vitamin D3 levels and liver dysfunction markers such as AST and INR, and a positive association with albumin levels, suggesting a strong link between Vitamin D3 status and liver synthetic function. Vitamin B12 levels also showed weak but significant positive correlations with AST and INR.

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