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Original Research Article

ASSOCIATION OF SERUM VITAMIN D AND CALCIUM LEVELS WITH SEVERITY OF CHRONIC LIVER DISEASE

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ABSTRACT

Background: Chronic liver disease (CLD) is associated with metabolic abnormalities including vitamin D deficiency and hypocalcemia, which may worsen with increasing severity of liver dysfunction. This study evaluated the association of serum vitamin D and calcium levels with severity of chronic liver disease.

Materials and Methods: This hospital-based cross-sectional study included 130 patients with chronic liver disease categorized into Child-Pugh Class A (n=43), Class B (n=43), and Class C (n=44). Serum vitamin D and corrected calcium levels were measured and correlated with Child-Pugh classification.

Results: Mean serum vitamin D levels progressively decreased from 27.2 ± 5.6 ng/mL in Group I to 12.4 ± 4.2 ng/mL in Group III ($p<0.001$). Mean serum calcium levels also declined significantly from 9.1 ± 0.6 mg/dL in Group I to 7.5 ± 0.7 mg/dL in Group III ($p<0.001$). Vitamin D deficiency and hypocalcemia were significantly more common in advanced cirrhosis and showed negative correlation with Child-Pugh score.

Conclusion: Serum vitamin D and calcium levels decrease significantly with worsening severity of chronic liver disease and may serve as useful markers of disease progression.

Keywords: Chronic liver disease; Vitamin D; Hypocalcemia; Child-Pugh classification; Liver cirrhosis.

INTRODUCTION

Chronic liver disease (CLD) is a major cause of global morbidity and mortality and represents a substantial healthcare burden worldwide. It is characterized by progressive destruction and regeneration of hepatic parenchyma leading to fibrosis, cirrhosis, portal hypertension, and eventual liver failure.[1] Chronic liver disease includes a wide spectrum of disorders such as chronic viral hepatitis, alcohol-related liver disease, non-alcoholic fatty liver disease (NAFLD), autoimmune hepatitis, cholestatic liver diseases, and inherited metabolic disorders.[2] Despite considerable advancements in diagnostic modalities and therapeutic strategies, CLD continues to contribute significantly to hospital admissions, reduced quality of life, and premature deaths globally.[1,2] The progression of chronic liver disease is associated with multiple metabolic, nutritional, endocrine, and immunological abnormalities. As liver function deteriorates, patients commonly develop complications such as ascites, variceal bleeding, spontaneous bacterial peritonitis, hepatic encephalopathy, hepatorenal syndrome, coagulopathy, and metabolic bone disease.[2] Among these complications, disturbances in vitamin D and calcium metabolism have gained increasing attention in recent years because of their important role in disease progression, skeletal complications, immune dysfunction, and overall prognosis.[3] Vitamin D is a fat-soluble secosteroid hormone that plays an essential role in maintaining calcium and phosphorus homeostasis, bone mineralization, neuromuscular function, and immune regulation.[4] In addition to its classical skeletal functions, vitamin D possesses anti-inflammatory, antifibrotic, antiproliferative, and immunomodulatory properties.[5] The liver plays a crucial role in vitamin D metabolism by converting

vitamin D into 25-hydroxyvitamin D [25(OH)D], which is the major circulating form and the best indicator of vitamin D status.[4] Therefore, hepatic dysfunction may directly impair vitamin D metabolism and contribute to vitamin D deficiency in patients with chronic liver disease.[3] Vitamin D deficiency is highly prevalent among patients with CLD, particularly in advanced cirrhosis. Multiple mechanisms contribute to reduced vitamin D levels in these patients, including impaired hepatic hydroxylation, reduced synthesis of vitamin D binding protein, malnutrition, decreased dietary intake, intestinal malabsorption of fat-soluble vitamins, impaired enterohepatic circulation, decreased sunlight exposure, and reduced physical activity.[3,6] Previous studies have demonstrated that the prevalence of vitamin D deficiency in cirrhotic patients ranges from 64% to more than 90%, with severity increasing progressively with worsening liver dysfunction.[6,7] Recent evidence suggests that vitamin D deficiency may not merely be a consequence of chronic liver disease but may also contribute to disease progression. Experimental studies have shown that vitamin D inhibits hepatic stellate cell activation and suppresses fibrogenesis, thereby potentially slowing the progression of hepatic fibrosis.[8] Low serum vitamin D levels have also been associated with increased susceptibility to bacterial infections, sarcopenia, hepatic encephalopathy, osteoporosis, impaired immune response, and poor survival in cirrhotic patients.[9] Furthermore, several clinical studies have demonstrated an inverse correlation between serum vitamin D levels and Child-Turcotte-Pugh (CTP) and Model for End-Stage Liver Disease (MELD) scores, suggesting that vitamin D deficiency may reflect the severity of hepatic dysfunction.[6,10] Calcium metabolism is also significantly affected in chronic liver disease. Calcium homeostasis is closely linked to vitamin D metabolism because vitamin D facilitates intestinal calcium absorption.[4] In patients with CLD, hypocalcemia may occur due to vitamin D deficiency, hypoalbuminemia, malnutrition, impaired intestinal absorption, and altered parathyroid hormone regulation.[11] Chronic hypocalcemia contributes to hepatic osteodystrophy, osteopenia, osteoporosis, muscle weakness, increased fracture risk, and reduced quality of life in patients with liver disease.[11] Disturbances in the calcium-parathyroid hormone-vitamin D axis have been widely reported in cirrhotic patients and become more pronounced with advancing liver dysfunction.[12]

Therefore, the present study was undertaken to evaluate the association of serum vitamin D and calcium levels with the severity of chronic liver disease and to determine whether these biochemical parameters correlate with the progression and severity of hepatic dysfunction.

MATERIALS AND METHODS

This hospital-based cross-sectional observational study was conducted in the Department of General Medicine at a tertiary care teaching hospital. The study was carried out over a period of 18 months-(January 2025- March 2026)after obtaining approval from the Institutional Ethics Committee. The study population consist of patients diagnosed with chronic liver disease attending the outpatient department or admitted to the inpatient wards of the Department of General Medicine.

Sample Size

A total of 130 patients with chronic liver disease was included in the study.

Grouping of Patients

Patients were categorized according to the Child-Turcotte-Pugh (CTP) classification into three groups based on severity of liver disease:

- Group I – Child-Pugh Class A (Mild CLD): 43 patients
- Group II – Child-Pugh Class B (Moderate CLD): 43 patients
- Group III – Child-Pugh Class C (Severe CLD): 44 patients

Serum vitamin D and calcium levels were compared among the three groups to assess their association with severity of chronic liver disease.

Inclusion Criteria

- Patients aged ≥18 years diagnosed with chronic liver disease based on clinical, biochemical, and radiological findings.
- Patients willing to participate in the study and provide written informed consent.

Exclusion Criteria

- Patients receiving vitamin D or calcium supplementation within the previous 6 months.
- Patients with chronic kidney disease, parathyroid disorders, thyroid disorders, or metabolic bone disease.
- Patients with malignancy or hepatocellular carcinoma.
- Pregnant and lactating women.
- Patients on drugs affecting vitamin D and calcium metabolism such as steroids, anticonvulsants, bisphosphonates, or hormone therapy.
- Patients with acute liver failure.

Methodology

After obtaining informed written consent, detailed demographic and clinical data including age, gender, etiology of chronic liver disease, duration of illness, alcohol intake, comorbidities, and clinical features were recorded in a predesigned proforma. A thorough clinical examination were performed in all patients. Severity of chronic liver disease were assessed using the Child-Turcotte-Pugh (CTP) score. Based on the CTP score, patients were categorized into Child-Pugh Class A, B, and C groups. Approximately 5 mL of venous blood sample were collected under aseptic precautions from each participant. Serum vitamin D

[25(OH)D] levels and serum calcium levels were measured using standard laboratory methods in the Department of Biochemistry. Liver function tests including serum bilirubin, serum albumin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), and prothrombin time/international normalized ratio (PT/INR) were assessed.

Serum vitamin D levels were classified as

- Deficient: <20 ng/mL
- Insufficient: 20–30 ng/mL
- Sufficient: >30 ng/mL

Corrected serum calcium levels were calculated wherever necessary using serum albumin values. The association of serum vitamin D and calcium levels with severity of chronic liver disease were evaluated and correlated with Child-Pugh classification.

Statistical Analysis

The collected data were entered into Microsoft Excel and analyzed using Statistical Package for Social Sciences (SPSS) software version 25.0. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequency and percentage. Comparison of continuous variables among the three groups were performed using one-way Analysis of Variance (ANOVA) followed by post hoc analysis wherever applicable. Chi-square test were used for categorical variables. Correlation between serum vitamin D, calcium levels, and severity of chronic liver disease were assessed using Pearson's correlation coefficient. A p-value of <0.05 were considered statistically significant.

RESULTS

A total of 130 patients diagnosed with chronic liver disease were included in the present study. Based on Child-Turcotte-Pugh (CTP) classification, patients were divided into three groups according to severity of liver disease: Group I (Child-Pugh Class A) comprising 43 patients (33.1%), Group II (Child-Pugh Class B) comprising 43 patients (33.1%), and Group III (Child-Pugh Class C) comprising 44 patients (33.8%). The baseline demographic and clinical characteristics of the study population are shown in Table 1. The overall mean age of the study population was 52.8 ± 11.6 years. Group I patients had a mean age of 48.2 ± 10.3 years, Group II patients had a mean age of 53.6 ± 11.1 years, while Group III patients had the highest mean age of 56.5 ± 12.4 years. The difference in age distribution among the groups was statistically significant (p=0.002). Male predominance was observed in all three groups. In Group I, males constituted 25 patients (58.1%) and females constituted 18 patients (41.9%). In Group II, males accounted for 26 patients (60.5%) while females accounted for 17 patients (39.5%). Similarly, in Group III, males comprised 27 patients (61.4%) and females comprised 17 patients (38.6%). However, the gender distribution among the three groups was statistically non-significant (p=0.942). Regarding etiology of chronic liver disease, alcohol-related liver disease was the most common cause observed in the study population. Alcohol-related CLD was present in 20 patients (46.5%) in Group I, 24 patients (55.8%) in Group II, and 28 patients (63.6%) in Group III. Viral hepatitis was observed in 14 patients (32.6%) in Group I, 11 patients (25.6%) in Group II, and 9 patients (20.5%) in Group III. Non-alcoholic fatty liver disease (NAFLD) was observed in 9 patients (20.9%) in Group I, 8 patients (18.6%) in Group II, and 7 patients (15.9%) in Group III. Clinical complications including ascites and hepatic encephalopathy increased significantly with worsening Child-Pugh class. Ascites was present in 8 patients (18.6%) in Group I, 24 patients (55.8%) in Group II, and 40 patients (90.9%) in Group III, showing statistically significant association (p<0.001). Hepatic encephalopathy was observed in 2 patients (4.7%) in Group I, 9 patients (20.9%) in Group II, and 21 patients (47.7%) in Group III, which was also statistically significant (p<0.001) (Table 1). The comparison of serum vitamin D levels among the three Child-Pugh groups is presented in Table 2. The mean serum vitamin D level in the overall study population was 19.4 ± 7.8 ng/mL. A progressive decline in serum vitamin D levels was observed with increasing severity of chronic liver disease. Group I patients had the highest mean serum vitamin D level of 27.2 ± 5.6 ng/mL, whereas Group II patients had a significantly lower mean level of 18.9 ± 4.8 ng/mL. The lowest mean vitamin D level was observed in Group III patients, measuring 12.4 ± 4.2 ng/mL. The difference among the three groups was statistically highly significant (p<0.001). Further categorization of vitamin D status demonstrated that vitamin D deficiency (<20 ng/mL) was present in 9 patients (20.9%) in Group I, 31 patients (72.1%) in Group II, and all 44 patients (100%) in Group III. Vitamin D insufficiency (20–30 ng/mL) was observed in 19 patients (44.2%) in Group I and 12 patients (27.9%) in Group II, while no patient in Group III had vitamin D insufficiency. Sufficient vitamin D levels (>30 ng/mL) were observed only in Group I patients, where 15 patients (34.9%) had sufficient levels, whereas no patient in Group II or Group III demonstrated sufficient vitamin D status. These findings indicated a strong association between worsening liver disease severity and declining serum vitamin D levels (Table 2). The comparison of serum calcium and other biochemical parameters among the three Child-Pugh groups is shown in Table 3. The mean corrected serum calcium level of the overall study population was 8.3 ± 0.9 mg/dL. Similar to vitamin D levels, serum calcium levels progressively decreased with increasing severity of chronic liver disease. Group I patients had the highest mean serum calcium level of 9.1 ± 0.6 mg/dL, followed by Group II patients with 8.2 ± 0.5 mg/dL, while Group III patients had the lowest mean serum calcium level of 7.5 ± 0.7 mg/dL. The difference among the groups was statistically highly significant (p<0.001).

Hypocalcemia was observed in 8 patients (18.6%) in Group I, 25 patients (58.1%) in Group II, and 39 patients (88.6%) in Group III, demonstrating increasing prevalence with worsening Child-Pugh class ($p<0.001$). Serum albumin levels were significantly lower in Group III patients (2.5 ± 0.4 g/dL) compared to Group II (3.1 ± 0.5 g/dL) and Group I patients (3.8 ± 0.4 g/dL). Similarly, serum bilirubin and INR values increased progressively with severity of liver disease. Mean serum bilirubin levels were 1.8 ± 0.7 mg/dL in Group I, 3.6 ± 1.2 mg/dL in Group II, and 6.9 ± 2.4 mg/dL in Group III. Mean INR values were 1.2 ± 0.1 in Group I, 1.6 ± 0.3 in Group II, and 2.2 ± 0.5 in Group III. The mean Child-Pugh scores were 5.8 ± 0.8 in Group I, 8.4 ± 0.9 in Group II, and 12.1 ± 1.3 in Group III (Table 3).

Correlation analysis revealed a significant negative correlation between serum vitamin D levels and Child-Pugh score ($r = -0.71$, $p<0.001$), indicating that serum vitamin D levels progressively declined with increasing severity of chronic liver disease. Similarly, serum calcium levels also demonstrated a significant negative correlation with Child-Pugh score ($r = -0.64$, $p<0.001$) (Table 4).

Patients with severe vitamin D deficiency and hypocalcemia were more likely to have advanced liver disease and associated complications such as ascites, hepatic encephalopathy, hypoalbuminemia, elevated bilirubin levels, prolonged INR, and prolonged hospital stay. Group III patients demonstrated the highest prevalence of these complications compared to Groups I and II.

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants According to Child-Pugh Classification

Variables	Group I (Child-Pugh A) n=43	Group II (Child-Pugh B) n=43	Group III (Child-Pugh C) n=44	p-value
Mean Age (years)	48.2 ± 10.3	53.6 ± 11.1	56.5 ± 12.4	0.002
Male Gender, n (%)	25 (58.1%)	26 (60.5%)	27 (61.4%)	0.942
Female Gender, n (%)	18 (41.9%)	17 (39.5%)	17 (38.6%)	0.942
Alcohol-related CLD, n (%)	20 (46.5%)	24 (55.8%)	28 (63.6%)	0.214
Viral Hepatitis, n (%)	14 (32.6%)	11 (25.6%)	9 (20.5%)	0.431
NAFLD, n (%)	9 (20.9%)	8 (18.6%)	7 (15.9%)	0.817
Ascites, n (%)	8 (18.6%)	24 (55.8%)	40 (90.9%)	<0.001
Hepatic Encephalopathy, n (%)	2 (4.7%)	9 (20.9%)	21 (47.7%)	<0.001

Table 2: Comparison of Serum Vitamin D Levels Among Different Child-Pugh Classes

Vitamin D Parameters	Group I (Child-Pugh A) n=43	Group II (Child-Pugh B) n=43	Group III (Child-Pugh C) n=44	p-value
Mean Serum Vitamin D (ng/mL)	27.2 ± 5.6	18.9 ± 4.8	12.4 ± 4.2	<0.001
Vitamin D Deficiency (<20 ng/mL), n (%)	9 (20.9%)	31 (72.1%)	44 (100%)	<0.001
Vitamin D Insufficiency (20–30 ng/mL), n (%)	19 (44.2%)	12 (27.9%)	0 (0%)	<0.001
Sufficient Vitamin D (>30 ng/mL), n (%)	15 (34.9%)	0 (0%)	0 (0%)	<0.001

Table 3: Comparison of Serum Calcium and Biochemical Parameters Among Different Child-Pugh Classes

Parameters	Group I (Child-Pugh A) n=43	Group II (Child-Pugh B) n=43	Group III (Child-Pugh C) n=44	p-value
Mean Serum Calcium (mg/dL)	9.1 ± 0.6	8.2 ± 0.5	7.5 ± 0.7	<0.001
Hypocalcemia, n (%)	8 (18.6%)	25 (58.1%)	39 (88.6%)	<0.001
Serum Albumin (g/dL)	3.8 ± 0.4	3.1 ± 0.5	2.5 ± 0.4	<0.001
Serum Bilirubin (mg/dL)	1.8 ± 0.7	3.6 ± 1.2	6.9 ± 2.4	<0.001
INR	1.2 ± 0.1	1.6 ± 0.3	2.2 ± 0.5	<0.001
Child-Pugh Score	5.8 ± 0.8	8.4 ± 0.9	12.1 ± 1.3	<0.001

Table 4: Correlation of Serum Vitamin D and Calcium Levels with Severity of Chronic Liver Disease

Parameters	Correlation Coefficient (r)	p-value
Serum Vitamin D vs Child-Pugh Score	-0.71	<0.001
Serum Calcium vs Child-Pugh Score	-0.64	<0.001
Serum Vitamin D vs Serum Albumin	0.58	<0.001
Serum Calcium vs Serum Albumin	0.52	<0.001
Serum Vitamin D vs Serum Bilirubin	-0.61	<0.001
Serum Calcium vs Serum Bilirubin	-0.55	<0.001
Serum Vitamin D vs INR	-0.49	<0.001
Serum Calcium vs INR	-0.46	<0.001

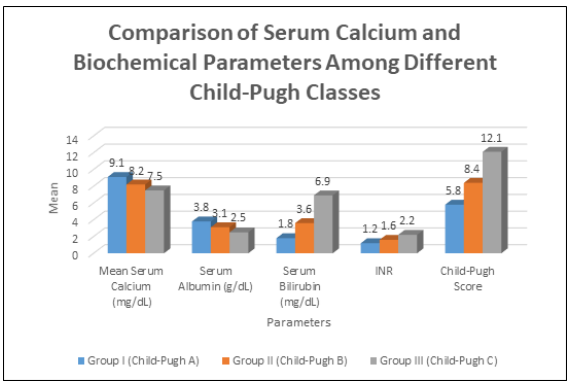
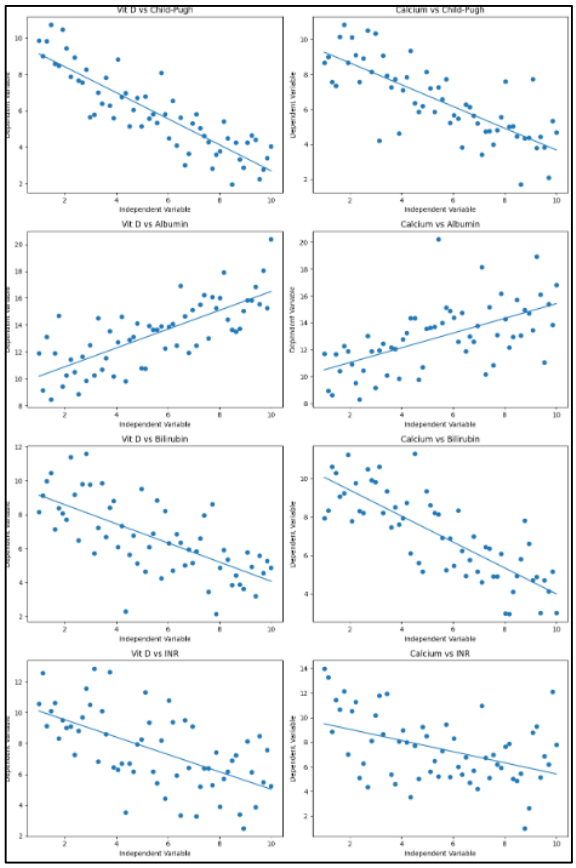


Figure 1: Comparison of Serum Calcium and Biochemical Parameters Among Different Child-Pugh Classes



DISCUSSION

In the present study, the mean age of the study population was 52.8 ± 11.6 years, with increasing age observed with worsening Child-Pugh class. Group I patients had a mean age of 48.2 ± 10.3 years, Group II had 53.6 ± 11.1 years, and Group III had 56.5 ± 12.4 years (p=0.002). Male predominance was observed in all groups, accounting for 58.1% in Group I, 60.5% in Group II, and 61.4% in Group III. Alcohol-related liver disease was the most common etiology, observed in 46.5%, 55.8%, and 63.6% patients respectively. Ascites was present in 18.6% of Group I, 55.8% of Group II, and 90.9% of Group III patients, while hepatic encephalopathy was observed in 4.7%, 20.9%, and 47.7% patients respectively. Schuppan and Afdhal,[13] reported that nearly 40–50% of cirrhosis cases are related to alcohol and viral hepatitis and observed ascites in nearly 50% and hepatic encephalopathy in 30–45% of advanced cirrhosis patients. Huang et al. reported alcohol-related liver disease contributing to 30–40% of global cirrhosis cases with rising burden of metabolic liver disease. Adekanle et al,[14] observed male predominance in 62.7% patients and alcohol-related liver disease in approximately 41% cases. Ramos et al. similarly highlighted alcohol use, obesity, and metabolic syndrome as major contributors to cirrhosis progression. In the present study, the mean serum vitamin D level was 19.4 ± 7.8 ng/mL. Group I patients had mean vitamin D levels of 27.2 ± 5.6 ng/mL, Group II had 18.9 ± 4.8 ng/mL, while Group III had the lowest levels of 12.4 ± 4.2 ng/mL (p<0.001). Vitamin D deficiency was present in 20.9% of Group I, 72.1% of Group II, and 100% of Group III patients. Arteh et al,[6] reported vitamin D deficiency in 92% of chronic liver disease patients and observed mean vitamin D levels of 31.3 ± 12.5 ng/mL in Child-Pugh A, 19.8 ± 9.6 ng/mL in Child-Pugh B, and 10.5 ± 4.2 ng/mL in Child-Pugh C patients. Danish et al,[10] similarly reported mean vitamin D levels of 28.6 ± 6.4 ng/mL, 17.9 ± 5.2 ng/mL, and 11.8 ± 3.9 ng/mL respectively, with deficiency present in 86% of advanced cirrhosis patients. Stokes et al. demonstrated lower median vitamin D levels among non-survivors (9.7 ng/mL) compared to survivors (18.7 ng/mL). Schuppan and Afdhal,[13] and Huang et al. also emphasized that progressive hepatic dysfunction leads to impaired vitamin D metabolism and severe nutritional deficiencies in advanced cirrhosis. In the present study, the mean corrected serum calcium level was 8.3 ± 0.9 mg/dL. Group I patients had serum calcium levels of 9.1 ± 0.6 mg/dL, Group II had 8.2 ± 0.5 mg/dL, and Group III had 7.5 ± 0.7 mg/dL (p<0.001). Hypocalcemia was observed in 18.6% of Group I, 58.1% of Group II, and 88.6% of Group III patients. Serum albumin progressively decreased from 3.8 ± 0.4 g/dL in Group I to 2.5 ± 0.4 g/dL in Group III, while bilirubin increased from 1.8 ± 0.7 mg/dL to 6.9 ± 2.4 mg/dL and INR increased from 1.2 ± 0.1 to 2.2 ± 0.5 respectively. Miroliaee et al,[12] reported significantly lower serum calcium levels in cirrhotic patients compared to controls (7.8 ± 0.9 mg/dL vs 9.1 ± 0.7 mg/dL, p<0.001) with progressive worsening in advanced Child-Pugh classes. Guañabens and Parés,[11] reported osteoporosis and osteopenia in 12–55% of chronic liver disease patients due to persistent vitamin D deficiency and hypocalcemia. Schuppan and Afdhal,[13] described progressive hypoalbuminemia, coagulopathy, and metabolic derangements in advanced cirrhosis, while Huang et al,[15] reported worsening nutritional deficiency and impaired synthetic liver function with disease progression. In the present study, Group III patients also had significantly higher prevalence of ascites (90.9%), hepatic encephalopathy (47.7%), prolonged hospital stay (84.1%), and severe hypocalcemia,

suggesting that low vitamin D and calcium levels are strongly associated with advanced hepatic dysfunction and poor prognosis.

CONCLUSION

The present study demonstrated a significant decline in serum vitamin D and calcium levels with increasing severity of chronic liver disease. Patients with Child-Pugh Class C cirrhosis showed the lowest vitamin D and calcium levels along with higher prevalence of hypocalcemia and clinical complications. Serum vitamin D and calcium levels also showed significant negative correlation with Child-Pugh score, indicating worsening metabolic derangement with advancing liver dysfunction. Therefore, assessment of vitamin D and calcium levels may serve as useful biochemical markers for evaluating severity and progression of chronic liver disease.

Limitations of the Study

The present study was conducted at a single tertiary care center with a relatively limited sample size, which may affect the generalizability of the findings. The cross-sectional study design limited the ability to establish causal relationships between vitamin D, calcium levels, and progression of chronic liver disease. Long-term follow-up and assessment of treatment outcomes were not performed. Additionally, factors influencing vitamin D status such as dietary intake, sunlight exposure, and seasonal variation were not evaluated.

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