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Investigation of 25-Hydroxyvitamin D Levels in Patients with Cirrhosis: Relationship with Liver Disease Severity, Etiology, and Complications

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ABSTRACT

Introduction: Vitamin D deficiency is frequently observed in people with cirrhosis, underscoring the liver's essential role in metabolizing vitamin D. This research sought to assess 25-hydroxyvitamin D [25(OH)D] levels in individuals with cirrhosis and to evaluate the association between these levels and disease extent, etiologies, and cirrhosis-related complications.

Methods: This study included 118 adult patients with cirrhosis who were evaluated at the Gastroenterology Department of Bezmialem Vakıf University. Participants ranged in age from 18 to 90 years. The study excluded individuals who were pregnant; had taken vitamin D supplements in the past six months; or had non- hepatocellular carcinoma malignancies, hematologic cancers, metabolic bone diseases, chronic kidney disease, parathyroid disorders, had undergone thyroidectomy, or other chronic conditions affecting vitamin D metabolism. The severity of liver disease was assessed using the Child-Turcotte-Pugh (Child-Pugh) classification and the Model for End-Stage Liver Disease (MELD) score. Data were examined with SPSS version 20.0.

Results: The average age of the patients was 58.7±11.8 years, with 56.8% being male. Cirrhosis was attributed to cryptogenic causes in 53.4% of cases, to hepatitis B virus in 28.80%, to hepatitis C virus in 8.50%, to alcohol in 7.60%, and to drugs in 1.70%. A deficiency in vitamin D was detected in 93.2% of the patients; 5.10% had insufficient levels, and only 1.70% had normal levels. Serum 25(OH)D levels demonstrated a positive correlation with albumin (p=0.003), total cholesterol (p=0.035), and triglycerides (p=0.006), and were negatively correlated with prothrombin time (p=0.003), international normalized ratio (p=0.011), Child-Pugh score (p=0.008), and MELD score (p=0.045). The levels of vitamin D did not significantly differ based on the cause of cirrhosis or the presence of cirrhosis-related complications.

Conclusion: Vitamin D insufficiency is a frequent problem among individuals with cirrhosis, regardless of its underlying cause. Reduced vitamin D levels are related to increasingly severe liver disease and to reduced liver function, rather than to the cause or complications of cirrhosis.

Keywords: 25-hydroxyvitamin D, cirrhosis, Child-Pugh score, MELD, liver disease severity

Introduction

Cirrhosis marked by extensive liver scarring, the development of regenerative nodules, and a progressive deterioration in liver function (1-3). It remains a key contributor to global morbidity and mortality and is associated with numerous complications (4,5).

Vitamin D, serves a vital role not solely in preserving calcium balance and bone health but also in modulating the immune system, cell growth, differentiation, and fibrogenesis (6,7). The liver is crucial for vitamin D metabolism, as it performs 25-hydroxylation and produces vitamin D-binding proteins. Therefore, it is biologically plausible that individuals with persistent liver disease may exhibit from vitamin D deficiency (6,8). Vitamin D deficiency is also extremely common among those with persistent liver disease and cirrhosis, with prevalence rates ranging from 64% to 92% (9-11).

Several factors have been identified as potential contributors to the low vitamin D levels observed in individuals with cirrhosis, including limited sunlight exposure, insufficient dietary intake, absorption issues, impaired liver hydroxylation, reduced production of vitamin D-binding proteins, and increased vitamin breakdown (8,12). Furthermore, vitamin D insufficiency may contribute to the progression of hepatic fibrosis, a diminished answer to antiviral therapy in chronic hepatitis C, an increased susceptibility to infections, and a higher risk of mortality

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(13-15). However, it remains unclear whether vitamin D insufficiency primarily reflects causation of cirrhosis, liver dysfunction, or complications associated with cirrhosis.

Methods

The study, which was retrospective and descriptive, examined 118 adult patients with cirrhosis evaluated at the Gastroenterology Department of Bezmialem Vakıf University between 2012 and 2016. The participants, aged 18-90 years, had chronic liver disease, including cirrhosis, and met the criteria for inclusion in the study.

Individuals between the ages of 18 and 90 with chronic liver disease or cirrhosis were considered eligible. Exclusion criteria were: pregnancy; use of vitamin supplements within the previous 6 months; non-hepatocellular carcinoma (HCC) solid malignancy; hematologic malignancy; history of metabolic bone disease; chronic kidney disease; hyperparathyroidism; hypoparathyroidism; prior thyroidectomy; and any other chronic disease that could affect vitamin D levels.

Ethics

Authorization for this research was received from the Ethics Committee of Bezmialem Vakıf University (approval number: 20/274, date: 07.11.2017). The study's retrospective nature allowed for the waiver of informed consent.

Laboratory Analysis

Venous blood samples were collected and centrifuged at 2000 × g for 10 minutes to obtain plasma. The concentration of 25-hydroxyvitamin D [25(OH)D] 3 in the blood was measured using a validated kit on a Shimadzu LC-20AT instrument. According to the Endocrine Society, vitamin D levels are classified as: deficiency,<20 ng/mL; insufficiency, 20-30 ng/mL; and normal, >30 ng/mL (16).

Clinical and Laboratory Variables

Data on patient demographics, the causes of cirrhosis, and associated complications were gathered from hospital records. The laboratory tests included measurements of glucose, white blood cell count (WBC), high-density lipoprotein (HDL), platelet count (PLT), hemoglobin (HGB), creatinine, lactate dehydrogenase (LDH), albumin, C-reactive protein (CRP), low-density lipoprotein (LDL), triglycerides, erythrocyte sedimentation rate (ESR), total cholesterol, HbA1c, ferritin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), prothrombin time (PT), and international normalized ratio (INR) (Table 1).

Cirrhosis-related complications included decompensation, ascites, spontaneous ascitic infection, hepatic encephalopathy, variceal bleeding, hepatorenal syndrome, portal vein thrombosis (PVT), hepatopulmonary syndrome, HCC, portal hypertension, and esophageal varices.

Assessment of Liver Disease Severity

The extent of the disease was measured using the Model for End-Stage Liver Disease (MELD) and Child-Turcotte-Pugh (Child-Pugh) scoring systems. The Child-Pugh classification was noted as stage A, B, or C (17).

Table 1. Baseline hematological and biochemical characteristics of the patients

Parameter	Mean ± SD	Median	Minimum	Maximum
Leukocyte (×10³/µL)	4.5±1.7	4.3	1.2	9.3
Hemoglobin (g/dL)	11.5±2.2	11.7	6.8	16.7
Platelet (×10³/µL)	103±59	88	27	352
Glucose (mg/dL)	118±42	106	55	325
Creatinine (mg/dL)	0.8±0.6	0.7	0.4	7.4
AST (U/L)	59.8±48.7	41.5	3	298
ALT (U/L)	47.9±48.0	32	6	266
ALP (U/L)	119.9±55.9	106	39	347
GGT (U/L)	88±133	54	8	1257
LDH (U/L)	215±65	202	62	524
25 (OH)D (ng/mL)	10.9±9.4	9.6	4	89
Total bilirubin (mg/dL)	1.4±1.6	1.0	0.2	13.0
Albumin (g/dL)	3.5±0.6	3.6	1.3	4.8
Prothrombin time (s)	17.4±3.8	16.5	11.4	32.0
INR	1.3±0.3	1.3	0.9	2.7
Total cholesterol (mg/dL)	160±56	156	53	384
Triglycerides (mg/dL)	119±77	95	33	456
LDL cholesterol (mg/dL)	98±43	97	13	251
HDL cholesterol (mg/dL)	40.9±18.7	38.5	3	87
C-reactive protein (mg/L)	0.7±1.8	0.3	0	19
HbA1c (%)	5.9±1.3	5.4	4.3	10.6
Erythrocyte sedimentation rate (mm/h)	22.9±16.1	18	3	88
Ferritin (ng/mL)	119±224	37.5	4	1649

SD: Standard deviation, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, ALP: Alkaline phosphatase, GGT: Gamma-glutamyl transferase, LDH: Lactate dehydrogenase, INR: International normalized ratio, ESR: Erythrocyte sedimentation rate, HbA1c: Hemoglobin A1c, 25(OH)D: 25-hydroxyvitamin D

Statistical Analysis

We used SPSS version 20.0 to analyze the data. We showed continuous data mean ± standard deviation or median (range), based on how they were spread out. We presented categorical data as numbers and percentages. We assessed the data distribution visually and using statistical tests. To compare two groups, we used the independent samples t-test or the Mann-Whitney U test, depending on which was appropriate. For comparisons involving three or more groups, we used one-way analysis of variance or the Kruskal-Wallis test. We used Pearson or Spearman correlation tests. A p value less than 0.05 was deemed statistically significant.

Results

Baseline Characteristics

Overall, 118 patients with cirrhosis were included in the analysis. The average age was 58.7±11.8 years (median, 59 years; range, 25-85 years). Of the patients, 67 (56.8%) were male and 51 (43.2%) were female.

The average MELD score was 9.5±2.7 and the average Child-Pugh score was 7.2±2.4. According to Child-Pugh stage, 62 patients (52.5%) were stage A, 31 (26.3%) stage B, and 25 (21.2%) stage C (Table 2).

The etiologies of cirrhosis were cryptogenic in 63 patients (53.4%), hepatitis B virus in 34 (28.8%), hepatitis C virus in 10 (8.5%), alcohol-related in 9 (7.6%), and drug-related in 2 (1.7%).

Regarding complications, portal hypertension was present in 93.2% of patients; esophageal varices in 86.4% of patients; decompensated cirrhosis in 51.7% of patients; ascites in 39.8% of patients; variceal bleeding in 31.4% of patients; hepatic encephalopathy in 25.4% of patients; PVT in 22.0% of patients; hepatorenal syndrome in 18.6% of patients; ascitic infection in 11.0% of patients; HCC in 11.0% of patients; and hepatopulmonary syndrome in 1.7% of patients (Table 3).

The mean serum vitamin D level was 10.9±9.4 ng/mL. Vitamin D deficiency was detected in 110 patients (93.2%), insufficiency in 6 patients (5.1%), and normal vitamin D levels in only 2 patients (1.7%).

When patients were stratified by Child-Pugh stage, vitamin D insufficiency was present in 91.9% of stage A patients, 96.8% of stage B patients, and 92.0% of stage C patients.

Vitamin D Levels According to Cirrhosis Etiology

Mean vitamin D levels according to etiology were as follows: cryptogenic cirrhosis, 12.6±11.7 ng/mL; hepatitis B-related cirrhosis, 9.3±5.3 ng/mL; hepatitis C-related cirrhosis, 7.5±3.6 ng/mL; alcohol-related cirrhosis, 9.9±5.4 ng/mL; and drug-related cirrhosis, 6.9±4.1 ng/mL. No statistically significant difference was found. In serum vitamin D levels among the different etiologic subgroups (p=0.210).

Vitamin D Levels and Cirrhosis-Related Complications

No notable association identified between vitamin D concentrations and decompensated cirrhosis (p=0.621), ascites (p=0.233), hepatic encephalopathy (p=0.215), variceal bleeding (p=0.988), ascitic infection (p=0.071), hepatorenal syndrome (p=0.670), hepatopulmonary syndrome (p=0.706), PVT (p=0.988), HCC (p=0.463), portal hypertension (p=0.427), or esophageal varices (p=0.794) (Table 4).

Vitamin D Levels and Laboratory Parameters

No statistically significant difference was found.among genders, with men having 10.3±6.8 ng/mL and women 11.4±10.9 ng/mL (p=0.511).

Table 2. Distribution of Child-Pugh and MELD scores of the patients

A) Continuous variables				
Parameter	**Mean ± SD**	**Median**	**Min**	**Max**
MELD score	9.5±2.7	9	6	20
Child-Pugh score	7.2±2.4	6	5	15
B) Categorical variables				
Parameter	**Category**	**n**	**%**	
Child-Pugh class	A	62	52.5	
	B	31	26.3	
	C	25	21.2	

SD: Standard deviation, MELD: Model for End-Stage Liver Disease, Min: Minimum, Max: Maximum, Child-Pugh: Child-Turcotte-Pugh

Additionally, vitamin D levels showed no significant correlation with variables such as age, WBC, HGB, PLT, glucose, creatinine, LDH, LDL, CRP, HDL, ESR, HbA1c, ferritin, GGT, AST, ALP, ALT, or bilirubin (Table 5).

Serum 25(OH)D levels displayed a positive correlation with albumin (rho: 0.271, p=0.003), total cholesterol (rho: 0.195, p=0.035), and triglycerides (rho: 0.251, p=0.006). In contrast, vitamin D levels were negatively correlated with PT (rho: -0.237, p=0.003) and INR (rho: -0.234, p=0.011).

Vitamin D Levels and Hepatic Disease Severity

Serum 25(OH)D levels were negatively associated with Child-Pugh score (rho: -0.243, p=0.008) and MELD score (rho: -0.185, p=0.045) (Table 6).

According to Child-Pugh stage, mean vitamin D levels were 11.2±6.5 ng/mL in stage A, 11.7±14.9 ng/mL in stage B, and 9.1±6.2 ng/mL in stage C. Although vitamin D levels tended to decrease with advancing Child-

Table 3. Distribution of cirrhosis-related complications

Parameter	Category	n	%
Cirrhosis status	Compensated	57	48.3
	Decompensated	61	51.7
Ascites	None	71	60.2
	Mild	15	12.7
	Moderate	8	6.8
	Severe	24	20.3
Ascitic infection	Present	13	11.0
	Absent	105	89.0
Hepatic encephalopathy	Present	30	25.4
	Absent	88	74.6
Encephalopathy stage	Stage 1–2	21	17.8
	Stage 3–4	9	7.6
Variceal bleeding	Present	37	31.4
	Absent	81	68.6
Esophageal varices	None	16	13.6
	Grade 1	34	28.8
	Grade 2	36	30.5
	Grade 3	32	27.1
Band ligation	Present	26	22.0
	Absent	92	78.0
Hepatorenal syndrome	Present	22	18.6
	Absent	96	81.4
Hepatopulmonary syndrome	Present	2	1.7
	Absent	116	98.3
Portal vein thrombosis	None	92	78.0
	Partial	24	20.3
	Complete	2	1.7
Hepatocellular carcinoma	Present	13	11.0
	Absent	105	89.0
Portal hypertension	None	8	6.8
	Stage 1–2	101	85.6
	Stage 3–4	9	7.6

PVT: Portal vein thrombosis, HCC: Hepatocellular carcinoma

Table 4. Vitamin D Levels According to cirrhosis-related complications

Complication	Category	Mean ± SD (ng/mL)	p value
Decompensated cirrhosis	Present	10.0±5.4	0.621
	Absent	11.8±12.2	
Ascites	None	10.8±6.3	0.233
	Mild	15.9±20.0	
	Moderate	7.9±4.5	
	Severe	9.1±5.7	
Hepatic encephalopathy	None	11.4±10.3	0.215
	Stage 1–2	10.4±6.0	
	Stage 3–4	7.2±3.6	
Variceal bleeding	Absent	11.3±10.8	0.988
	Present	10.1±5.1	
Ascitic infection	Absent	11.3±9.7	0.071
	Present	7.9±4.5	
Hepatorenal syndrome	Absent	11.1±10.0	0.670
	Present	10.0±5.8	
Hepatopulmonary syndrome	Absent	10.9±9.4	0.706
	Present	13.0±11.5	
Portal vein thrombosis	None	11.1±10.1	0.988
	Partial	9.9±4.9	
	Complete	16.0±16.9	
Hepatocellular carcinoma	Absent	10.9±9.8	0.463
	Present	10.7±5.0	
Portal hypertension	None	8.1±4.1	0.427
	Stage 1–2	11.3±9.9	
	Stage 3–4	8.7±4.9	
Esophageal varices	None	10.3±6.7	0.794
	Grade 1	11.2±6.4	
	Grade 2	12.4±14.6	
	Grade 3	9.3±4.4	
Band ligation	Absent	11.3±10.3	0.560
	Present	9.5±4.8	

Statistical analysis: Mann–Whitney U test and Kruskal–Wallis test were used as appropriate. SD: Standard deviation

Pugh stage, the difference among Child-Pugh stages failed to achieve statistical significance (p=0.140).

Discussion

This study found that over 90% of individuals with cirrhosis experienced vitamin D deficiency. Additionally, lower vitamin D levels were linked to signs of reduced hepatic synthetic function and increased disease severity.

These findings are consistent with previous studies indicating a elevated prevalence of inadequate vitamin D levels in those with cirrhosis (9,10,18). Our findings reveal that levels are more closely linked to the extent of hepatic dysfunction than to the underlying cause of cirrhosis. Earlier studies have also observed that vitamin D deficiency correlates

Table 5. Correlation between vitamin D levels and liver function tests

Parameter	Correlation coefficient (Rho)	p value
AST (U/L)	0.007	0.940
ALT (U/L)	0.007	0.937
ALP (U/L)	0.007	0.939
GGT (U/L)	0.092	0.322
Total bilirubin (mg/dL)	-0.080	0.390
Prothrombin time (s)	-0.237	0.003
INR	-0.234	0.011

Analysis: Spearman correlation. AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, ALP: Alkaline phosphatase, GGT: Gamma-glutamyl transferase, INR: International normalized ratio

Table 6. Correlation between vitamin D levels and Child–Pugh and MELD scores

Parameter	Correlation coefficient (Rho)	p value
Child–Pugh score	-0.243	0.008
MELD score	-0.185	0.045

Analysis: Spearman correlation. MELD: Model for End-Stage Liver Disease

with disease severity rather than its etiology (11,19). The observed associations between vitamin D and albumin, PT, and INR further support the idea that a decline in liver synthetic capacity plays a critical role in the development of vitamin D deficiency. the liver is responsible for both hydroxylating vitamin D and producing binding proteins, impaired liver function could directly result in lower vitamin D levels in the blood (6,8,20). Thus, vitamin D may serve as an indirect marker of liver reserve.

While some research has indicated a link between low serum vitamin D levels and outcomes such as infection or decompensation (11,14,15), the literature is inconsistent. The lack of correlation in our study may be due to its retrospective design, modest sample size, and variation in complication profiles. The positive relationship between vitamin D and lipid parameters, including cholesterol and triglycerides, should also be considered in the context of liver function. Lower lipid levels are commonly found in progressive liver disease due to impaired synthesis (21-24). Therefore, this relationship likely reflects overall hepatic dysfunction rather than a direct metabolic interaction.

Vitamin D deficiency in cirrhosis is likely multifactorial, involving reduced sun exposure, nutritional deficiencies, impaired intestinal absorption, decreased hepatic conversion, and altered metabolism (8,20,25-27). Besides being a marker of disease severity, vitamin D may also contribute to disease progression through its roles in inflammation, fibrosis, and immune regulation (13,28-30).

Study Limitations

The present study has several limitations. First, its retrospective and observational design precludes establishing causal relationships between vitamin D levels and liver disease severity. Second, this single-center study had a relatively small sample size, which may limit the generalizability of the findings. Third, longitudinal follow-up data were unavailable;

therefore, the prognostic significance of vitamin D levels over time could not be evaluated. Fourth, the unequal distribution of certain cirrhosis-related complications may have limited the statistical power of subgroup analyses. In addition, seasonal variations in sunlight exposure, which may influence serum 25(OH)D concentrations, could not be taken into account because the timing of vitamin D measurements was not standardized. Multivariable analyses were not performed; therefore, the potential confounding effects could not be fully adjusted for.

Conclusion

Vitamin D deficiency is highly prevalent among patients with cirrhosis. Lower serum 25(OH)D levels were associated with greater liver disease severity and impaired hepatic synthetic function, rather than with the etiology or specific complications of cirrhosis. These findings suggest that vitamin D deficiency may represent a marker of disease severity in cirrhosis. However, given the observational nature of this study, these associations should not be interpreted as causal relationships. Further prospective studies are warranted to determine whether correction of vitamin D deficiency may influence clinical outcomes.

Ethics

Ethics Committee Approval: Authorization for this research was received from the Ethics Committee of Bezmialem Vakıf University (approval number: 20/274, date: 07.11.2017).

Informed Consent: The study's retrospective nature allowed for the waiver of informed consent.

Footnotes

Authorship Contributions: Concept - A.Ö., A.T.İ.; Design - A.Ö., A.T.İ.; Data Collection or Processing - A.Ö.; Analysis or Interpretation - A.Ö., A.T.İ.; Literature Search - A.Ö., A.T.İ.; Writing - A.Ö.

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