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Original Research Article

Study on Vitamin D Assay in Chronic Non Cholestatic Liver Disease

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Abstract

Introduction: Vitamin D is a secosteroid hormone with pleiotropic actions that extend beyond skeletal homeostasis to immunomodulatory, anti-inflammatory and anti-fibrotic effects—pathways that are highly relevant to chronic liver disease (CLD) of non-cholestaticaetiology, including metabolic dysfunction-associated steatotic liver disease (MASLD/NAFLD), alcohol-related liver disease (ALD) and chronic viral hepatitis.

Aims and Objectives: To assay vitamin D in patients with non-cholestatic chronic liver disease and to compare the parameters of liver function test (LFT) with vitamin D levels and correlate the two if possible.

Materials and Methods: The present study was a descriptive observational study with cross sectional design. This Study was conducted over 1 year period from the date of approval of protocol at Nadia district hospital, Krishnanagar, West Bengal.

Result: In this study of patients with chronic non-cholestatic liver disease, serum vitamin D status was associated with variations in liver function and disease etiology. While age, sex, and BMI did not differ significantly across vitamin D groups, anti-HCV positivity and underlying etiology showed significant associations, with alcohol-related liver disease more common in patients with lower vitamin D levels and NASH predominating in those with sufficient levels. Liver function parameters—including SGOT, SGPT, ALP, GGT, bilirubin, albumin, and globulin—differed significantly among the groups, indicating greater hepatic dysfunction in patients with vitamin D deficiency or insufficiency.

Conclusion: This study highlights a significant association between serum vitamin D status and both liver function and disease etiology in patients with chronic non-cholestatic liver disease. Patients with lower vitamin D levels tended to exhibit more pronounced alterations in liver function markers, including elevated liver enzymes and reduced serum albumin, suggesting greater hepatic dysfunction.

Keywords: Vitamin D, Chronic liver disease, Non-cholestatic liver disease, Liver function tests.

Introduction

Vitamin D is a secosteroid hormone with pleiotropic actions that extend beyond skeletal homeostasis to immunomodulatory, anti-inflammatory and anti-fibrotic effects—pathways that are highly relevant to chronic liver disease (CLD) of non-cholestaticaetiology, including metabolic dysfunction-associated steatotic liver disease (MASLD/NAFLD), alcohol-related liver disease (ALD) and chronic viral hepatitis. Hepatic 25-hydroxylation (principally via CYP2R1) generates 25-hydroxyvitamin D [25(OH)D], the accepted biomarker of vitamin D status, which circulates largely bound to vitamin D–binding protein and albumin; thus, hepatocellular dysfunction, reduced protein synthesis, malnutrition and limited sunlight exposure frequently converge to produce low total 25(OH)D in CLD [1,2]. Observational cohorts and reviews consistently report a high prevalence of vitamin D insufficiency/deficiency across CLD severities, with levels declining as fibrosis and synthetic failure progress, and inverse correlations with Child–Pugh and MELD scores have been described [1,3,4,5]. In NAFLD, low 25(OH)D has been associated with steatosis, necroinflammation and fibrosis in several populations, and Mendelian-randomisation analyses suggest an inverse relationship between genetically predicted vitamin D status and NAFLD risk, although causality for histological improvement remains debated [5,6]. Meta-analyses of supplementation trials in NAFLD demonstrate reliable repletion of serum 25(OH)D

and modest lipid profile benefits, but inconsistent effects on aminotransferases and glycaemic indices, underscoring biological plausibility without definitive therapeutic proof for liver endpoints [6]. In chronic hepatitis C, multiple studies report an association between low 25(OH)D and advanced fibrosis, with mechanistic work implicating vitamin D receptor signalling in stellate-cell activation and matrix remodelling [2,4]. Beyond fibrosis, deficiency has been linked to infections, hepatic encephalopathy and mortality in cirrhosis, suggesting prognostic value that may transcend bone outcomes [1,5]. These clinicopathological links raise important questions for laboratories and clinicians: which assay, what threshold, and in whom should we test? Assay methodology varies—automated competitive immunoassays, radioimmunoassay and liquid chromatography–tandem mass spectrometry (LC-MS/MS) are all used—with notable inter-method bias, variable cross-reactivity for 25(OH)D_2/D_3, and matrix effects; contemporary endocrine and laboratory consensus documents emphasisestandardisation (e.g., VDSP alignment) and recognise ongoing controversy around "optimal" cut-offs (20 vs 30 ng/mL) [7,8]. In cirrhosis, low albumin and binding-protein concentrations may disproportionately depress total 25(OH)D while free/bioavailable fractions remain less affected, a nuance that could influence interpretation when comparing across disease severities and assays [1,7]. Guideline perspectives have shifted: the 2024 Endocrine Society guideline advises against routine 25(OH)D screening for disease prevention in otherwise unselected populations, reflecting outcome-based evidence gaps; nonetheless, high-risk groups and established indications remain appropriate contexts for measurement and treatment [7].

For hepatology, where fracture risk, sarcopenia, infection susceptibility and encephalopathy carry prognostic weight, targeted assessment may still be clinically justified, particularly in decompensated disease, ALD, malnutrition, and pre-transplant evaluations [1,3,5,9,10]. Against this background, a focused "Study on Vitamin D Assay in Chronic Non-Cholestatic Liver Disease" is timely. It can quantify the burden of hypovitaminosis D by a standardised method, examine concordance or bias across commonly used assays, and explore correlations between 25(OH)D and validated measures of liver severity (e.g., fibrosis surrogates, Child–Pugh, MELD), nutrition and extra-hepatic outcomes. By integrating rigorous pre-analytical control (season, latitude, BMI, diabetes, alcohol and supplement use), method selection (preferably LC-MS/MS or a VDSP-aligned immunoassay), and clinically meaningful thresholds prespecified from consensus statements, such a study can clarify the interpretive landscape for hepatology services. Ultimately, delineating how assay choice and disease-related binding-protein perturbations shape 25(OH)D readouts will help determine when and how vitamin D testing should inform risk stratification and management in non-cholestatic CLD [1–10].

Materials and Methods

Study Area: Nadia district hospital, Krishnanagar, West Bengal.

Study Population: Patients with the diagnosis of non-cholestatic liver disease attending their department were included.

All the patients were explained in detail about the study, and an informed consent was taken.

Study Design: Descriptive observational study with cross sectional design.

Study Duration: 1 year period from the date of approval of protocol.

Inclusion Criteria: Detailed history and clinical evaluation done.

The inclusion criteria for the patients in this study were:

All the patients diagnosed with non cholestatic chronic liver disease diagnosed with the help of USG (ultrasonography) andLFT (Liver function test) (USG suggestive of chronic liver disease is increased echotexture and LFT with low albumin and mildly raised liver enzymes)

Exclusion Criteria

1. Patients with cholestatic liver disease
2. Patients who were on calcium supplements and vitamin D
3. Patients on medications which affect bone mineral density
4. Postmenopausal women
5. Patients with coexistent chronic kidney disease

Sample Size: 55 Patients with chronic non-cholestatic liver disease.

Study Tools

1. Pre designed and pre tested Questionnaire
2. USG machine
3. Relevant investigation records

Statistical Analysis: For statistical analysis, data were initially entered into a Microsoft Excel spreadsheet and then analysed using SPSS (version 27.0; SPSS Inc., Chicago, IL, USA) and GraphPad Prism (version 5). Numerical variables were summarized using means and standard deviations, while Data were entered into Excel and analyzed using SPSS and GraphPad Prism. Numerical variables were summarized using means and standard deviations, while categorical variables

were described with counts and percentages. Two-sample t-tests were used to compare independent groups, while paired t-tests accounted for correlations in paired data. Chi-square tests (including Fisher's exact test for small sample sizes) were used for categorical data comparisons. P-values ≤ 0.05 were considered statistically significant.

Result

Table 1: Distribution of Age and Sex across Serum Vitamin D Status in Chronic Non-Cholestatic Liver Disease Patients

		Serum Vitamin D Group				
		Deficiency	**Insufficiency**	**Sufficiency**	**Total**	**P-value**
Age in Group	≤50	2(6.3%)	1(6.3%)	0(0%)	3(5.5%)	0.2808
	51-60	2(6.3%)	5(31.3%)	0(0%)	7(12.7%)	
	61-70	11(34.4%)	4(25%)	4(57.1%)	19(34.5%)	
	71-80	10(31.3%)	5(31.3%)	2(28.6%)	17(30.9%)	
	81-90	7(21.9%)	1(6.3%)	1(14.3%)	9(16.4%)	
	Total	32(100%)	16(100%)	7(100%)	55(100%)	
Sex	Female	13(40.6%)	5(31.3%)	5(71.4%)	23(41.8%)	0.1944
	Male	19(59.4%)	11(68.8%)	2(28.6%)	32(58.2%)	
	Total	32(100%)	16(100%)	7(100%)	55(100%)	

Table 2: Distribution of Viral Serology and Etiology across Serum Vitamin D Status in Chronic Non-Cholestatic Liver Disease Patients

		Serum Vitamin D Group				
		Deficiency	**Insufficiency**	**Sufficiency**	**Total**	**P-value**
Anti HCV	Negative	32(100%)	12(75.0%)	7(100%)	51(92.7%)	0.0052
	Positive	0(0%)	4(25%)	0(0%)	4(7.3%)	
	Total	32(100%)	16(100%)	7(100%)	55(100%)	
Hepatitis B serology	Negative	31(96.9%)	16(100%)	7(100%)	54(98.2%)	0.6935
	Positive	1(3.1%)	0(0%)	0(0%)	1(1.8%)	
	Total	32(100%)	16(100%)	7(100%)	55(100%)	
Etiology	Alcohol	12(37.5%)	12(75%)	0(0%)	24(43.6%)	<0.0001
	Hepatitis C related	1(3.1%)	4(25%)	0(0%)	5(9.1%)	
	Nash related	19(59.4%)	0(0%)	7(100%)	26(47.3%)	
	Total	32(100%)	16(100%)	7(100%)	55(100%)	

Table 3: Comparison of Demographic, Anthropometric, and Biochemical Parameters across Vitamin D Status in Chronic Non-Cholestasis Liver Disease Patients

		Number	**Mean**	**SD**	**Minimum**	**Maximum**	**Median**	**p-value**
Age (yr)	Deficiency	32	71.4375	10.4633	48	90	72.5	0.2825
	Insufficiency	16	66.6875	9.4919	49	81	67.5	
	Sufficiency	7	71	6.4031	62	82	70	
BMI (kg/m2)	Deficiency	32	27.9219	2.4033	24.5	32	27	0.4288
	Insufficiency	16	27.0625	2.3585	25.5	33	26.5	
	Sufficiency	7	28	0	28	28	28	
Size of portal vein (in mm)	Deficiency	32	15.6406	0.7853	14	16.5	16	<0.0001
	Insufficiency	16	14.5	0.8944	14	16	14	
	Sufficiency	7	16	0	16	16	16	
Serum vitamin D(OH) assay (ng/ml)	Deficiency	32	13.8	3.3884	3	19	15	<0.0001
	Insufficiency	16	22.675	1.2969	20.5	23.4	23.4	
	Sufficiency	7	32	0	32	32	32	
Total bilirubin (mg/dl)	Deficiency	32	3.0494	1.1174	1.6	4.22	3.1	<0.0001
	Insufficiency	16	1.7175	0.0939	1.56	1.77	1.77	
	Sufficiency	7	2.3	0	2.3	2.3	2.3	

Table 4: Comparison of Liver Function Parameters across Vitamin D Status Categories in Chronic Non-Cholestatic Liver Disease Patients

		Number	Mean	SD	Minimum	Maximum	Median	p-value
SGOT(U/L)	Deficiency	32	68.4375	25.3885	25	111	67	<0.0001
	Insufficiency	16	140.75	23.7023	101	154	154	
	Sufficiency	7	35	0	35	35	35	
SGPT (U/L)	Deficiency	32	37.7188	10.5195	25	65	34	<0.0001
	Insufficiency	16	102.5	13.4164	80	110	110	
	Sufficiency	7	27	0	27	27	27	
ALP(U/L)	Deficiency	32	65.25	18.2968	45	90	66	<0.0001
	Insufficiency	16	101.75	22.8079	89	140	89	
	Sufficiency	7	78	0	78	78	78	
Albumin (g/dl)	Deficiency	32	2.8625	0.3867	2.1	3.5	2.9	0.0034
	Insufficiency	16	3.25	0.4472	3	4	3	
	Sufficiency	7	3.2	0	3.2	3.2	3.2	
Globulin(g/dl)	Deficiency	32	4.3469	0.4174	3.5	4.9	4.4	0.0004
	Insufficiency	16	3.925	0.1342	3.7	4	4	
	Sufficiency	7	4.1	0	4.1	4.1	4.1	

Table 5: Distribution of mean Gama GT(U/L): Serum vitamin D Group

		Number	Mean	SD	Minimum	Maximum	Median	p-value
Gama GT(U/L)	Deficiency	32	73.8125	22.3079	49.0000	110.0000	70.0000	<0.0001
	Insufficiency	16	122.2500	1.3416	120.0000	123.0000	123.0000	
	Sufficiency	7	60.0000	.0000	60.0000	60.0000	60.0000	

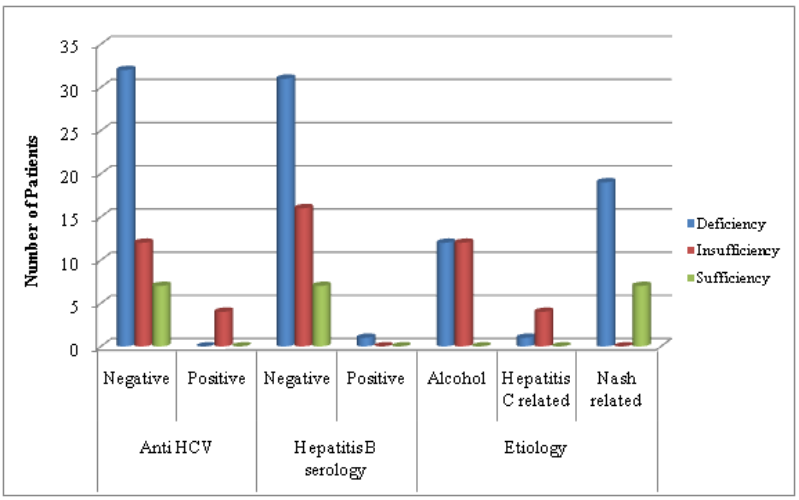

Figure 1: Distribution of Viral Serology and Etiology across Serum Vitamin D Status in Chronic Non-Cholestatic Liver Disease Patients

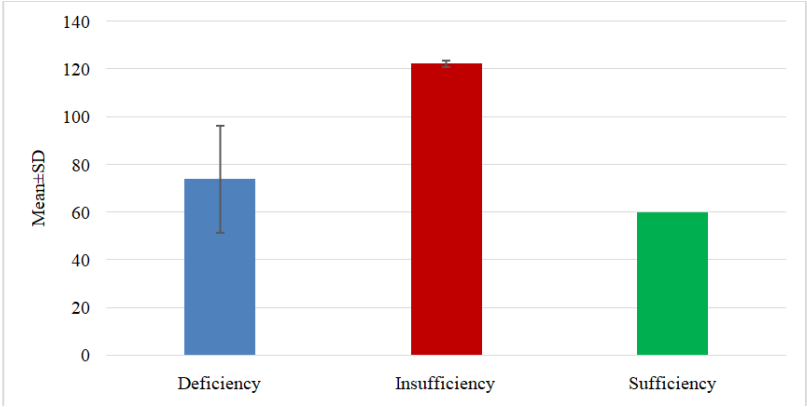

Figure 2: Distribution of mean Gama GT(U/L): Serum vitamin D Group

In this study of 55 patients with chronic non-cholestatic liver disease, the distribution of age across serum vitamin D groups showed that the majority of patients in the deficiency group were aged 61–70 years (34.4%) and 71–80 years (31.3%), while in the insufficiency group, most were between 51–60 years (31.3%) and 71–80 years (31.3%). In the sufficiency group, 57.1% were aged 61–70 years, with smaller proportions in the 71–80 (28.6%) and 81–90 years (14.3%) categories. The difference in age distribution among vitamin D groups was not statistically significant ($p = 0.2808$). Regarding sex distribution, males predominated in the deficiency (59.4%) and insufficiency (68.8%) groups, whereas females comprised a majority in the sufficiency group (71.4%). This difference was also not statistically significant ($p = 0.1944$).

Among the 55 patients, anti-HCV serology showed that all patients in the vitamin D deficiency (100%) and sufficiency (100%) groups were negative, while 75% of patients in the insufficiency group were negative and 25% were positive. This difference was statistically significant ($p = 0.0052$). For hepatitis B serology, nearly all patients were negative across all vitamin D groups, with only one patient (3.1%) in the deficiency group testing positive, showing no significant difference between groups ($p = 0.6935$). Regarding etiology, alcohol-related liver disease was predominant in the insufficiency group (75%) and common in the deficiency group (37.5%) but absent in the sufficiency group. Hepatitis C–related liver disease was observed in 25% of the insufficiency group and 3.1% of the deficiency group, while non-alcoholic steatohepatitis (NASH) was most frequent in the sufficiency group (100%) and in 59.4% of the deficiency group. These differences in etiology across vitamin D groups were highly significant ($p < 0.0001$).

In this study of 55 patients with chronic non-cholestatic liver disease, the mean age did not differ significantly across serum vitamin D groups, with the deficiency, insufficiency, and sufficiency groups having mean ages of 71.44 ± 10.46, 66.69 ± 9.49, and 71 ± 6.40 years, respectively ($p = 0.2825$). Similarly, body mass index (BMI) showed no significant differences between groups (27.92 ± 2.40 vs. 27.06 ± 2.36 vs. 28 ± 0 kg/m²; $p = 0.4288$). The size of the portal vein, however, varied significantly among the groups, with the deficiency group showing a mean diameter of 15.64 ± 0.79 mm, insufficiency 14.5 ± 0.89 mm, and sufficiency 16 ± 0 mm ($p < 0.0001$). Serum 25-hydroxyvitamin D levels differed significantly, as expected, with mean levels of 13.8 ± 3.39 ng/mL in the deficiency group, 22.68 ± 1.30 ng/mL in the insufficiency group, and 32 ± 0 ng/mL in the sufficiency group ($p < 0.0001$). Total bilirubin levels also demonstrated significant differences, with the deficiency group showing the highest mean value (3.05 ± 1.12 mg/dL), followed by the sufficiency (2.3 ± 0 mg/dL) and insufficiency groups (1.72 ± 0.09 mg/dL) ($p < 0.0001$).

The analysis of liver function parameters across serum vitamin D groups revealed significant differences among the deficiency, insufficiency, and sufficiency categories. Mean SGOT levels were highest in the insufficiency group (140.75 ± 23.70 U/L) compared to the deficiency (68.44 ± 25.39 U/L) and sufficiency groups (35 ± 0 U/L) ($p < 0.0001$). Similarly, SGPT levels were significantly elevated in the insufficiency group (102.5 ± 13.42 U/L) relative to the deficiency (37.72 ± 10.52 U/L) and sufficiency groups (27 ± 0 U/L) ($p < 0.0001$). ALP levels also differed markedly, with the insufficiency group showing the highest mean (101.75 ± 22.81 U/L), followed by sufficiency (78 ± 0 U/L) and deficiency groups (65.25 ± 18.30 U/L) ($p < 0.0001$). Serum albumin was significantly lower in the deficiency group (2.86 ± 0.39 g/dL) compared to the insufficiency (3.25 ± 0.45 g/dL) and sufficiency groups (3.2 ± 0 g/dL) ($p = 0.0034$). Conversely, globulin levels were higher in the deficiency group (4.35 ± 0.42 g/dL) than in the insufficiency (3.93 ± 0.13 g/dL) and sufficiency groups (4.1 ± 0 g/dL) ($p = 0.0004$).

The serum gamma-glutamyltransferase (GGT) levels differed significantly across the vitamin D groups. The mean GGT was highest in the insufficiency group (122.25 ± 1.34 U/L), followed by the deficiency group (73.81 ± 22.31 U/L), and lowest in the sufficiency group (60 ± 0 U/L) ($p < 0.0001$).

Discussion

In this cohort of 55 patients with chronic non-cholestatic liver disease, vitamin-D strata tracked clinically meaningful differences in virology, etiology, and liver biochemistry despite broadly similar age and BMI distributions. The strikingly higher anti-HCV positivity confined to the insufficiency group (25%) mirrors prior work showing that lower 25-hydroxyvitamin D (25[OH]D) levels in HCV are linked to greater inflammation, fibrosis, and poorer treatment response, suggesting that even “insufficient” (not frankly deficient) status may matter biologically in viral hepatitis [13,20]. Etiologic patterns also aligned with contemporary literature: alcohol-related disease clustered in lower vitamin-D states, consistent with high rates of hypovitaminosis D in ALD and its association with worse outcomes [19,20], whereas the 100% NASH proportion in the sufficiency group (with small n) contrasts with the usual inverse relationship between vitamin D and NAFLD risk/severity reported by several analyses—though discordant findings exist and

some studies find no histologic gradient by 25(OH)D, underscoring heterogeneity and potential confounding by adiposity, season, and sampling frame [17,18]. Biochemically, the insufficiency group showed the highest mean SGOT, SGPT, ALP, and GGT, while the deficiency group exhibited the lowest albumin and highest bilirubin—together echoing the broad association between low vitamin D and more advanced hepatic dysfunction noted across CLD (lower albumin, higher bilirubin, and worse Child-Pugh/MELD) [11,12,16].

Portal vein caliber differed across groups; although direct links between 25(OH)D and measured portal pressure are mixed, lower vitamin-D status has been tied to portal-hypertension complications and decompensation risks in cirrhosis, suggesting that vitamin D may be a marker—or modest mediator—of the pathobiology driving portal hypertension [12,14,15].

Overall, our pattern converges with "another author's" series—Arteh et al., who reported near-universal vitamin-D deficiency in CLD—while extending it by showing that gradients across deficiency/insufficiency/sufficiency map onto distinct virologic and biochemical profiles even when age/BMI are balanced [11]. Clinically, these data support routine vitamin-D assessment in CLD and targeted correction alongside disease-specific management, particularly for patients with viral hepatitis or alcohol-related disease, where low 25(OH)D tracks with adverse phenotypes and outcomes [13–16,19,20].

Conclusion

This study highlights a significant association between serum vitamin D status and both liver function and disease etiology in patients with chronic non-cholestatic liver disease. Patients with lower vitamin D levels tended to exhibit more pronounced alterations in liver function markers, including elevated liver enzymes and reduced serum albumin, suggesting greater hepatic dysfunction.

Additionally, vitamin D status appeared to correlate with the underlying cause of liver disease, with alcohol-related liver disease more common among those with lower vitamin D levels, while non-alcoholic fatty liver disease was predominant in patients with sufficient levels.

These findings underscore the potential role of vitamin D as a marker of liver health and disease severity, suggesting that assessment and correction of vitamin D deficiency may be an important consideration in the management of chronic liver disease. Overall, maintaining adequate vitamin D levels could have implications not only for general health but also for optimizing liver function and potentially mitigating disease progression.

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