Original Article

Assessment of Nutritional, Clinical and Thyroid Profile of Liver Cirrhosis Patients and Their Association with Severity of Liver Cirrhosis

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Abstract

Background: Cirrhosis represents the end stage of chronic liver disease (CLD), commonly complicated by malnutrition, sarcopenia, thyroid dysfunction, and micronutrient deficiencies. These factors may affect disease severity and prognosis but remain underexplored in the Indian population. The objective is to assess the nutritional, clinical and thyroid profiles of liver cirrhosis patients in a tertiary care center and to find their association with the severity of liver cirrhosis. **Material and Methods:** This cross-sectional study was conducted at a tertiary care hospital in North-western India. One hundred adult patients diagnosed with liver cirrhosis were assessed through clinical evaluation & laboratory tests (including liver function, thyroid profile, vitamin B12 and vitamin D levels), and classified as per severity of cirrhosis using CTP scores. **Results:** The mean age of the participants was 59.2 ± 8.46 years and 85% were male. The leading cause was alcoholic liver disease (46%) followed by viral hepatitis (26%). Most patients were in CTP Class C (60%). The most predominant symptoms were abdominal distension (80%) and anorexia (74%), while predominant clinical signs were ascites (69%), icterus (65%) and pallor (63%). A statistically significant association was found between advance CTP class and worsening liver function parameters: higher bilirubin, prolonged PT-INR, and lower albumin levels ($p < 0.001$). Serum TSH levels were significantly lower in patients with higher CTP classes ($p = 0.026$), while T3 and T4 levels showed no significant variation across CTP classes. Serum vitamin D levels declined significantly with increasing severity of cirrhosis ($p = 0.022$). In contrast, Serum vitamin B12 levels showed increasing trend with disease progression, though this association was not statistically significant ($p = 0.258$). **Conclusion:** Progression of liver cirrhosis is associated with reductions in TSH and vitamin D levels, along with an increase vitamin B12 levels, emphasizing their potential as indicators of disease severity. Regular assessment of nutritional and thyroid profiles in cirrhotic patients could improve patient management and clinical outcomes.

Keywords: Liver cirrhosis, Child-Turcotte-Pugh score, Chronic liver disease, Thyroid stimulating hormone.

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Introduction

Chronic liver disease (CLD) is characterized by the gradual decline of liver structure and function, often accompanied by complications of portal hypertension such as ascites, splenomegaly, gastrointestinal bleeding and hepatic encephalopathy. Cirrhosis, the end stage of chronic liver disease, is defined by regenerative nodules surrounded by fibrous tissue, caused by ongoing liver damage. This structural change increases portal pressure, finally leading to end-stage liver disease.[1,2] Cirrhosis is a significant and increasing global health problem, ranking as the 14th leading cause of death worldwide and 4th in Central Europe, causing about 1.03 million deaths annually.[3] In developed countries chronic hepatitis C, non-alcoholic fatty liver disease (NAFLD) and alcohol abuse are primary causes. Meanwhile, hepatitis B remains the leading cause in many Asian and sub-Saharan African countries. In India, cirrhosis is a major health problem, accounting for 259,749 deaths in 2017—2.95% of the country's total deaths—and contributing to 18.3% of global cirrhosis-related deaths.[4]

Liver disease also influences the metabolism of several hormones and micronutrients. Vitamin and trace element deficiencies are commonly found in individuals with liver cirrhosis, irrespective of its underlying cause. This is often due to impaired liver function, insufficient dietary intake, poor absorption, and increased breakdown of nutrients. Additionally, factors such as malabsorption, impaired digestion, and the use of diuretics further contribute to these micronutrient deficiencies.[5]

Vitamin D is synthesized predominantly in the liver and functions as an important secosteroid hormone with pleiotropic effects. While its key regulatory role in calcium and bone

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homeostasis is well established, recently there is increasing recognition that vitamin D also regulates cell proliferation and differentiation, and has immunomodulatory, anti-inflammatory and anti-fibrotic properties. These non-skeletal effects are relevant in the pathogenesis and treatment of many causes of chronic liver disease. Vitamin D deficiency is frequently present in chronic liver disease and may predict non-response to antiviral therapy in chronic hepatitis C. Vitamin D deficiency also closely relates to the severity of non-alcoholic fatty liver disease (NAFLD) and is implicated in the pathogenesis of insulin resistance, a key factor in the development of NAFLD.[6,7] Vitamin D deficiency is linked to more severe disease progression and increased mortality. Recent studies have shown that between 64% and 92% of patients with chronic liver conditions suffer from vitamin D insufficiency or deficiency, a rate notably higher than in the general population. Moreover, the severity of vitamin D deficiency tends to rise in parallel with the progression of liver disease.[8,9]

The observed decline in vitamin D levels with worsening liver disease likely involves multiple contributing factors, which may differ based on the specific liver condition. Potential mechanisms include reduced exposure to vitamin D sources (like diet and sunlight), impaired absorption from the intestines, decreased liver production of vitamin D-binding protein (DBP) and albumin, reduced liver-mediated conversion of vitamin D to its active form (25(OH)D), and increased breakdown of 25(OH)D.[9,10]

Vitamin B12, mainly presenting two forms in humans: 5′-deoxyadenosylcobalamine and methyl cobalamin, was reported to be correlated with hepatitis and cirrhosis.[11] In addition, vitamin B12 was served as a cofactor for methyl malonyl CoA mutase, which managed the rate of long-chain fatty Acyl-CoA enter into mitochondria and influences lipid metabolic pathways.[12] Vitamin B12, predominantly stored in the liver, is often elevated in cases of acute and chronic liver disease. This may be due to hepatocellular injury (excess release hypothesis) or reduced hepatic clearance (reduced clearance hypothesis), both of which suggest elevated B12 could serve as a marker of disease severity in chronic hepatitis and cirrhosis.[13]

The liver also plays a central role in thyroid hormone metabolism, including the conversion of thyroxine (T4) to the biologically active triiodothyronine (T3), synthesis of thyroid-binding proteins, and hormone degradation. Thus, liver dysfunction can lead to thyroid abnormalities, which are reported in 13% to 61% of liver cirrhosis patients.[14] Hypothyroidism is usually associated with higher Body mass index (BMI) and dyslipidaemia, both of which contribute to metabolic associated fatty liver disease (MAFLD) progression. Reduced thyroid hormone levels decrease lipid metabolism, increase hepatic inflammation through increased tumour necrosis factor-alpha (TNF-α) and leptin, and suppress adiponectin—all leading to hepatic fibrosis. Conversely, longstanding hyperthyroidism can lead to liver injury from increased metabolic demands and, in advance cases, right-sided heart failure due to thyrotoxicosis.[15]

To evaluate disease severity and predict outcomes in cirrhotic patients, the Modified Child-Turcotte-Pugh score remains one of the most widely used and validated clinical tools.[16]

Objectives: This study intended to evaluate the nutritional, clinical and thyroid profiles of liver cirrhosis patients in a tertiary care center, with a focus on the relationship between these factors and the severity of liver cirrhosis.

Materials and Methods

This cross-sectional study was conducted at a tertiary care center in North-western India. The study included adult patients aged 18 years and above, diagnosed with liver cirrhosis and visited the gastroenterology and medicine department (inpatient or outpatient). Patients with hepatocellular carcinoma, severe extrahepatic disorders, or connective tissue diseases were excluded from study. Informed consent was obtained from all participants.

Clinical Data Collection: Patients' demographic profiles, clinical history, and symptoms were recorded. Laboratory investigations including complete blood counts, liver function tests (LFT), renal function tests, blood glucose, thyroid function tests and serum vitamin B12 levels were performed by using standard methods, including chemiluminescence immunoassay (CLIA) for thyroid function and vitamin B12 levels. The severity of cirrhosis assesses by CTP score.

Statistical Analysis: Data were analysed by SPSS version 25.0. Categorical variables were presented as percentages and proportions, while quantitative data were expressed as means and standard deviations. Chi-square tests were used to examine associations between categorical variables and comparisons of continuous variables were made by ANOVA test.

Results

Demographic Characteristics: The study included 100 patients diagnosed with liver cirrhosis. The mean age was 59.2 years (± 8.46) and 85% were male. The leading causes of liver cirrhosis were alcoholic liver disease (46%) and viral hepatitis (26%) [Table 1].

Child-Turcotte-Pugh (CTP) Classification

Most patients were in the advanced stages of liver cirrhosis, with 60% classified as CTP class C and 19% as class B [Table 1].

Prevalence of Symptoms and Signs

The most predominant symptoms were abdominal distension (80%), loss of appetite (74%), yellowing of the skin or eyes (69%), and fatigue (58%). On physical examination, ascites (69%), jaundice (65%) and pallor (63%), were commonly recorded [Table 2].

Liver Function Tests

Liver cirrhosis severity (CTP class) was strongly linked to liver function markers. Higher CTP classes showed increased bilirubin, PT, and INR, and decreased albumin levels ($p < 0.001$).

Thyroid Function Tests

No significant variations in T3 and T4 levels were found across different CTP classes. However, TSH levels were notably lower in patients with more severe liver disease (CTP C) compared to those with less advanced stages ($p = 0.026$) [Table 3].

Vitamin D and B12 Levels

Vitamin D levels showed significant differences among CTP classes, decreasing from an average of 29.98 ± 12.87 ng/mL in

CTP A to 21.29 ± 12.46 ng/mL in CTP C. In contrast, vitamin B12 levels did not vary significantly between groups (p = 0.258), ranging from 356.38 ± 247.16 pg/mL in CTP A to 494.21 ± 331.60 pg/mL in CTP C.

Table 1: Demographic profile, etiology and CTP classification of study participants

Variables		N=100	In %
Gender	Male	85	85
	Female	15	15
Age groups	40-49 years	16	16
	50-59 years	32	32
	60-70 years	52	52
CTP class	CTP-A	21	21
	CTP-B	19	19
	CTP-C	60	60
Etiology	Alcoholic	46	46
	Hepatitis B	20	20
	MAFLD	15	15
	Hepatitis C	6	6
	Auto-immune	6	6
	Idiopathic	6	6
	Wilson disease	1	1

Table 2: Prevalence of sign and symptoms

Symptoms	Prevalence of symptom (%)	Signs	Prevalence of sign (%)
Abdominal distension	80	Ascites	69
Anorexia	74	Icterus	65
Jaundice	69	Pallor	63
Fatigue	58	Loss of body hair	44
Hematemesis	42	Dyspnoea	38
Vomiting	40	Pedal edema	38
Fever	33	Palmer erythema	27
Dizziness	27	Parotid enlargement	24
Altered sensorium	7		
Oliguria	6		

Table 3: Mean values of laboratory parameters according to CTP class

Parameter	CTP A	CTP B	CTP C	P value
Bilirubin (mg/dl)	1.32 ± 0.26	1.62 ± 0.2	4.11 ± 1.96	<0.001
Albumin (g/dl)	3.74 ± 0.1	2.98 ± 0.14	2.56 ± 0.4	<0.001
PT INR	1.48 ± 0.11	2.01 ± 0.13	3.1 ± 0.65	<0.001
Vitamin D (ng/ml)	29.98 ± 12.87	26.17 ± 12.95	21.29 ± 12.46	0.022
Vitamin B12 (pg/ml)	356.38 ± 247.16	461.8 ± 286.33	494.21 ± 331.6	0.258
T3 (ng/ml)	1.25 ± 0.36	1.46 ± 0.29	1.39 ± 0.39	0.181
T4 (mcg/ml)	8.81 ± 2.36	9.77 ± 2.41	10.41 ± 2.76	0.057
TSH (µIU/ml)	3.58 ± 0.95	2.86 ± 1.82	2.67 ± 1.21	0.026

Discussion

In our study, the majority of cirrhotic patients were between 60–70 years, followed by the 50–59 age group. This is consistent with finding of study by Guptha et al. (17), who noted a higher prevalence of CLD in those over 60. Higher age is a major risk factor, associated with poorer outcomes in advance stages of liver cirrhosis.

In our study, the majority of patients (60%) were classified as CTP class C, followed by 21% in grade A and 19% in grade B. This pattern was also observed in the study of Bhattarai et al,[18] who reported 63.1% in grade C. Similar distributions were also observed in the study of Bhattacharyya et al,[19] and Hajiani et al,[20] highlighting the dominance of advanced-stage cirrhosis in our study.

In our study, abdominal distension was predominant symptom of cirrhosis, occurring in 80% of patients, followed by anorexia (74%), jaundice (69%), fatigue (58%), and vomiting (40%). These findings closely relate to those reported by Bhattarai et al. in patients with chronic liver disease.[18] Ascites was the predominant clinical sign, seen in 69% of patients, followed by icterus in 65%. This is consistent with findings from Bhattarai et al., who reported similar rates of icterus and pallor, as well as Maskey et al., who also reported ascites and icterus as the most common signs.[18,21]

Our study showed that higher CTP grades in cirrhosis patients were significantly associated with increased bilirubin and PT-INR levels, and decreased serum albumin levels. These findings are consistent with Ahmed Z et al,[22] who also reported rising bilirubin and PT-INR with increasing liver cirrhosis severity. However, unlike their study, our study showed a significant decline in albumin levels with increasing CTP grade.

Our study showed a significant negative correlation between serum vitamin D levels and CTP grade. This finding is consistent with the finding of study by Adiri W.N. et al., who reported that lower vitamin D levels were associated with increased severity of liver cirrhosis, highlighting its potential role as both a prognostic indicator and a therapeutic target.[23] Similarly,

Paternostro R. et al. also reported that vitamin D levels decreasing progressively with more advanced CTP stages, correlating with the severity of liver cirrhosis.[24] The finding of study by Iruzubieta P. et al. also supports this, indicating that advanced liver cirrhosis impairs the hydroxylation of vitamin D and decrease the synthesis of albumin and vitamin D binding protein (DBP), all of which leads to lower serum 25(OH)D levels. Additionally, vitamin D deficiency in chronic liver disease may result from a combination of various factors including inadequate sun exposure, poor dietary intake, corticosteroid use, reduced cutaneous synthesis due to jaundice, and reduced intestinal absorption caused by intestinal edema from portal hypertension or cholestasis-related bile salt disruption.[25] Finding of study by Khan M.A. et al. showed a significantly lower vitamin D levels in chronic liver disease patients, but they did not find a statistically significant correlation with disease stage.[26]

Our study noted a trend of increasing levels of vitamin B12 with advancing liver disease; however, the difference was not statistically significant. These results are consistent with findings by Shugihara T. et al., who showed significantly higher mean serum B12 concentrations in patients with Child-Pugh C compared to those with chronic hepatitis, Child-Pugh A, and B. The increasing B12 levels in advanced liver disease is likely due to reduced hepatic uptake and storage, along with increased release from damaged hepatocytes.[13] Kumar G.P. et al. also noted that vitamin B12 levels increase with higher CTP grade (27). Joge N.P.'s study also showed a progressive increase in B12 levels with worsening liver cirrhosis. This increase may be due to hepatocellular damage, which causes leakage of vitamin B12 into the bloodstream, leading to increase serum levels despite intracellular deficiency. Furthermore, damage to liver tissue may disrupt the storage and binding of vitamin B12 with transcobalamin, contributing to the observed rise in circulating levels.[28]

Our study showed that TSH levels were significantly lower in patients with CTP grade C, though still within the normal range. This finding contrasts with the study by Punekar P et al., who found significantly decrease free triiodothyronine (FT3) and free thyroxine (FT4) levels in cirrhotic patients, with a significant increase in TSH levels compared to healthy controls.[14] The discrepancy between our study and Punekar et al.'s findings may be attributed to differences in patient populations, disease etiology, or the use of different diagnostic methods.

Conclusion

In conclusion, our study highlights the close connection between liver dysfunction and changes in metabolism, nutrition, and hormone levels in patients with liver cirrhosis. A significant decrease in vitamin D levels with increasing cirrhosis severity suggests its potential role as a prognostic marker, whereas elevated vitamin B12 levels, though not statistically significant, may reflect underlying liver dysfunction. Regular monitoring of vitamin B12, vitamin D3 and thyroid profile is important for the effective management of cirrhotic patients. Identifying and treating vitamin D deficiency and thyroid abnormalities may help to improve overall care, quality of life and clinical outcomes in patients with liver cirrhosis. Further studies are needed to better understand these associations and evaluate the benefits of targeted interventions.

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Conflicts of interest

There are no conflicts of interest.

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