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Original Research Article

Association Between Subclinical Hypothyroidism and Severity of Liver Cirrhosis: A Cross-sectional Study from a Tertiary Care Center in Western India

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Abstract:

Background: Thyroid dysfunction, particularly subclinical hypothyroidism, is frequently observed in patients with chronic liver disease, but its clinical relevance in cirrhosis remains uncertain.

Objective: To assess the prevalence of subclinical hypothyroidism in cirrhotic individuals with cirrhosis and how it relates to the severity of liver disease.

Methods: A cross-sectional study was conducted over 6 months at Vyas Medical College and Hospital, Jodhpur. One hundred patients with diagnosed liver cirrhosis were enrolled. Thyroid function tests were performed, a high TSH combined with normal free T4 was considered subclinical hypothyroidism. Severity of cirrhosis was graded using the CTP and MELD scores. Statistical analysis assessed the relationship between thyroid status and cirrhosis severity.

Results: Subclinical hypothyroidism was detected in 32% of patients. The prevalence increased with advancing cirrhosis (18% in CTP A, 33% in CTP B, and 46% in CTP C; p=0.02). Patients with subclinical hypothyroidism had higher mean MELD scores (18.2 ± 3.6 vs 14.5 ± 3.2, p<0.01) and more frequent complications such as ascites and hepatic encephalopathy.

Conclusion: Subclinical hypothyroidism is common among cirrhotic patients and correlates with disease severity. Routine thyroid function screening may aid in risk stratification and comprehensive management of cirrhosis.

Keywords: Subclinical Hypothyroidism, Liver Cirrhosis, Child–Turcotte–Pugh, MELD Score, Thyroid Dysfunction.

Introduction

Liver cirrhosis represents the common final pathway of numerous chronic liver diseases and remains a leading cause of morbidity and mortality worldwide. It is characterized by progressive fibrosis, architectural distortion, and portal hypertension, eventually leading to complications such as ascites, variceal bleeding, encephalopathy, and hepatocellular carcinoma. In India, cirrhosis is highly prevalent, largely due to alcohol-related disease, viral hepatitis, and non-alcoholic fatty liver disease. Assessing disease severity is central to prognosis and management, and tools such as the Child–Turcotte–Pugh (CTP) score and the Model for End-Stage Liver Disease (MELD) score are widely employed. Yet cirrhosis is not limited to hepatic dysfunction alone—it disrupts several extrahepatic systems, including the endocrine axis, particularly thyroid function.

The thyroid and liver maintain a close physiological relationship. The liver plays an essential role in the metabolism, conversion, and clearance of thyroid hormones, while thyroid hormones regulate hepatic blood flow, lipid metabolism, and biliary excretion. In cirrhosis, these interdependent mechanisms are

disturbed, and abnormal thyroid function tests are frequently observed. Elevated thyroid-stimulating hormone combined with normal thyroxine levels is known as subclinical hypothyroidism, is the most frequent alteration. Although often asymptomatic, it may have significant implications, with reported prevalence in cirrhotic populations ranging from 20% to 40%. Pathophysiological contributors include reduced deiodinase activity, impaired protein synthesis, and cytokine-mediated suppression of the hypothalamic-pituitary-thyroid axis.

Emerging evidence suggests that subclinical hypothyroidism correlates with the severity of cirrhosis. Several studies have reported higher prevalence of thyroid dysfunction in advanced disease, and patients with abnormal thyroid function tend to show higher MELD and CTP scores. Moreover, these patients are more likely to develop complications such as refractory ascites and encephalopathy, supporting the idea that thyroid dysfunction is not only a marker of disease severity but may also worsen clinical outcomes by aggravating metabolic and circulatory disturbances. This association has raised interest in considering thyroid function as an adjunctive marker of prognosis in chronic liver disease.

Despite these observations, regional data from India, particularly from Western states, remain limited. Variations in etiology, nutritional status, and access to healthcare may influence both the prevalence and consequences of thyroid abnormalities in cirrhosis. The current study, conducted at a tertiary center in Jodhpur, was designed to evaluate the frequency of subclinical hypothyroidism in cirrhotic patients and examine its relationship with disease severity. By correlating thyroid function with CTP and MELD scores, this work aims to provide evidence for integrating simple thyroid function testing into routine evaluation of cirrhosis, potentially improving risk stratification and patient care.

Methods

Study Design and Setting: A cross-sectional observational study was conducted at Vyas Medical College and Hospital, Jodhpur, over 6 months (April – October 2025).

Participants: One hundred adult patients (>18 years) with a confirmed diagnosis of liver cirrhosis based on clinical, biochemical, radiological, and/or histological findings were included. Patients with known thyroid disease, recent thyroid medication use, pituitary disorders, or acute critical illness were excluded.

Data Collection: Demographic data, etiology of cirrhosis, clinical features, and complications (ascites, variceal bleeding, encephalopathy) were recorded. Laboratory parameters included liver function tests, coagulation profile, renal function, and thyroid profile (TSH, free T4).

Definitions:

- Subclinical hypothyroidism: TSH >5.0 mIU/L with normal free T4.
- Cirrhosis severity: assessed by CTP classification and MELD score.

Statistical Analysis: Continuous variables were expressed as mean ± SD, categorical variables as percentages. Chi-square test was applied for categorical comparisons, and independent t-test for continuous variables. Correlation between thyroid status and MELD/CTP scores was analyzed using Pearson correlation. A p-value <0.05 was considered significant.

Results

Baseline Characteristics:

Patient Characteristics: A total of 100 patients with cirrhosis were included. The mean age was 49.6 ± 11.2 years, and 68% were male. The leading etiologies were alcohol-related liver disease (42%), viral hepatitis (34%), and non-alcoholic steatohepatitis (16%). Baseline demographic and clinical variables were broadly similar between patients with normal thyroid function and those with subclinical hypothyroidism.

Prevalence of Subclinical Hypothyroidism: Subclinical hypothyroidism (SCH) was identified in 32 of 100 patients (32%). The prevalence of SCH rose progressively with increasing severity of cirrhosis. Among patients classified as Child–Turcotte–Pugh (CTP) class A, 18% had SCH, compared with 33% in class B and 46% in class C ($p = 0.02$).

Association with MELD Score: Patients with SCH had significantly higher MELD scores compared with euthyroid patients (18.2 ± 3.6 vs 14.5 ± 3.2, $p < 0.01$). The difference persisted after adjusting for age and sex. A positive correlation was observed between TSH levels and MELD scores ($r = 0.41$, $p < 0.01$), suggesting that worsening thyroid function reflects progressive hepatic impairment.

Clinical Complications: Patients with SCH experienced more frequent cirrhosis-related complications. Ascites was present in 78% of SCH patients compared with 52% in euthyroid patients ($p = 0.01$). Hepatic encephalopathy occurred in 28% versus 14%, respectively ($p = 0.04$). No significant differences were observed in rates of variceal bleeding or spontaneous bacterial peritonitis.

Table 1: Baseline characteristics of study population

Variable	Euthyroid (n=68)	SCH (n=32)	p-value
Age (years)	48.9 ± 10.7	51.2 ± 11.9	0.34
Male sex (%)	67.6	68.8	0.91
Alcoholic etiology (%)	41.1	43.7	0.79
Viral hepatitis (%)	35.2	31.2	0.68
NASH (%)	15.0	18.7	0.62

Table 2: Association between thyroid status and cirrhosis severity

Severity Measure	Euthyroid (n=68)	SCH (n=32)	p-value
Mean MELD score	14.5 ± 3.2	18.2 ± 3.6	<0.01
CTP A (%)	28	18	
CTP B (%)	36	33	
CTP C (%)	36	46	0.02

Table 3: Frequency of complications

Complication	Euthyroid (%)	SCH (%)	p-value
Ascites	52	78	0.01
Encephalopathy	14	28	0.04
Variceal bleeding	19	22	0.74
SBP	8	12	0.53

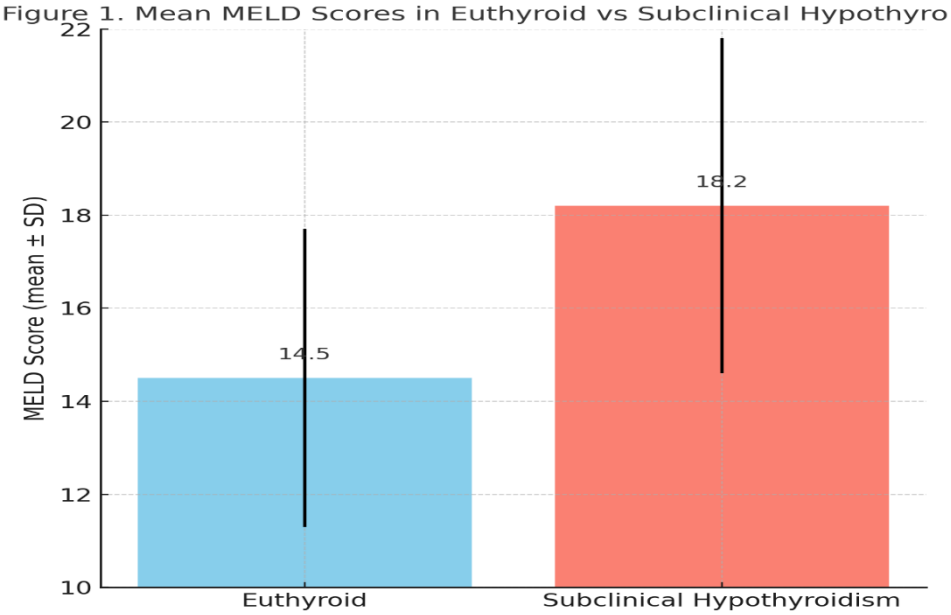

Figure 1: Mean MELD Scores in Subclinical Hypothyroidism vs Euthyroid

Discussion

The present study investigated the frequency of subclinical hypothyroidism among patients with cirrhosis and examined its association with the severity of liver disease. Nearly one-third of patients were found to have elevated TSH with normal thyroxine levels, highlighting that thyroid dysfunction is not an uncommon finding in cirrhotic populations. More importantly, the prevalence of subclinical hypothyroidism increased progressively with advancing Child–Turcotte–Pugh (CTP) stage, and patients with thyroid dysfunction had higher MELD scores than those with normal thyroid function. These findings demonstrate a close relationship between thyroid status and the degree of hepatic decompensation.

Our results are in line with prior reports from both Indian and international cohorts. Earlier studies have shown that between 20% and 40% of cirrhotics develop subclinical hypothyroidism, with the highest frequency in those with advanced disease. Bal and colleagues from India reported a prevalence of 29%, while Eshraghian et al. observed a rate of 33% in an Iranian cohort. Giannini and others demonstrated that thyroid abnormalities correlated with decompensated cirrhosis, with low T3 and elevated TSH levels serving as markers of poor prognosis. The similarity of our results with these

studies strengthens the evidence that thyroid dysfunction is a common and clinically relevant feature of cirrhosis.

The mechanisms underlying this association are multifactorial. The liver regulates thyroid hormone metabolism through deiodination, binding protein synthesis, and clearance pathways. As cirrhosis progresses, these processes become impaired. Reduced activity of type I deiodinase leads to diminished conversion of thyroxine (T4) to triiodothyronine (T3), while hypoalbuminemia reduces transport capacity for thyroid hormones. Cytokine-mediated suppression of the hypothalamic–pituitary–thyroid axis further disrupts normal regulation. Together, these alterations create a state of compensated thyroid dysfunction, in which TSH rises in an attempt to maintain circulating hormone levels. This explains why subclinical hypothyroidism becomes more prevalent as liver disease severity increases.

Beyond being a biochemical marker, subclinical hypothyroidism may contribute directly to complications of cirrhosis. In our study, patients with thyroid dysfunction had a higher frequency of ascites and encephalopathy. Hypothyroidism is known to impair renal sodium handling, reduce cardiac output, and increase systemic vascular resistance, all of which may exacerbate fluid retention and portal hypertension. Similarly, thyroid hormones play an important role in cerebral metabolism and neurotransmission, and thyroid dysfunction may lower the threshold for hepatic encephalopathy in vulnerable patients. These findings indicate that thyroid dysfunction may not simply reflect disease severity but may also accelerate decompensation.

The association between subclinical hypothyroidism and higher MELD scores in our study also deserves emphasis. Patients with thyroid dysfunction had mean MELD values nearly four points higher than those with normal thyroid function. Since the MELD score incorporates bilirubin, INR, and creatinine, it reflects both hepatic and systemic function. Thyroid dysfunction may worsen renal and circulatory status, indirectly influencing MELD parameters. Cakir et al. previously demonstrated that subclinical hypothyroidism predicted poorer survival in cirrhotic patients, mediated partly by higher MELD scores. This supports the idea that thyroid status could serve as a complementary prognostic indicator in chronic liver disease.

From a clinical standpoint, the findings of this study underscore the importance of routinely assessing thyroid function in cirrhotic patients. TSH and free T4 measurements are inexpensive and widely available. Their inclusion in baseline and follow-up evaluations could help identify patients at higher risk of complications and poor outcomes. Although the benefit of thyroid hormone replacement in subclinical cases remains uncertain, recognition of thyroid dysfunction allows clinicians to tailor monitoring and consider closer follow-up. In resource-limited settings where access to advanced imaging and transplantation is restricted, simple laboratory markers such as TSH may provide valuable additional prognostic information.

This study has several strengths, including its systematic evaluation of thyroid function, use of two established scoring systems for cirrhosis severity, and analysis of common complications. However, limitations should be acknowledged. The cross-sectional design prevents conclusions about causality, and follow-up data on survival or the impact of thyroid correction were not available. The study was also confined to a single center, which may limit generalizability. Despite these limitations, the findings add to the growing body of literature emphasizing the interplay between thyroid function and liver disease progression. Future longitudinal and interventional studies are needed to determine whether thyroid dysfunction independently predicts mortality and whether correction of subclinical hypothyroidism improves outcomes in cirrhotic patients.

Conclusion

In this study of 100 patients with liver cirrhosis, subclinical hypothyroidism was observed in almost one-third of cases and showed a clear association with advanced disease. Its frequency increased across Child–Turcotte–Pugh stages, and affected patients demonstrated higher MELD scores than those with normal thyroid function. Subclinical hypothyroidism was also linked to a greater incidence of complications such as ascites and encephalopathy, indicating that altered thyroid function may reflect both the severity and clinical expression of cirrhosis. These findings support incorporating thyroid function testing into the routine evaluation of cirrhotic patients as a simple and inexpensive marker of risk. While the prognostic implications appear significant, the therapeutic role of thyroid hormone supplementation in this context is uncertain. Larger, prospective studies are required to determine whether early recognition and management of subclinical hypothyroidism can modify outcomes and improve care in patients with chronic liver disease.

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