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Original Research Article

A STUDY OF THYROID DYSFUNCTION IN LIVER CIRRHOSIS AND ITS ASSOCIATION WITH SEVERITY OF LIVER CIRRHOSIS

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ABSTRACT

Background: Liver cirrhosis is associated with progressive hepatic dysfunction and metabolic disturbance. This study aimed to evaluate thyroid hormone levels and determine their association with the severity of liver cirrhosis using the Model for End-Stage Liver Disease (MELD) score. **Materials and Methods:** This hospital-based observational study was conducted at the Trichy SRM Medical College Hospital and Research Centre over six months. A total of 186 adult patients with confirmed liver cirrhosis were enrolled. Serum FT3, FT4, and TSH were measured, and the MELD score was calculated using bilirubin, creatinine, and INR values. **Results:** Among the 186 patients, 142 (76.3%) were men. Alcohol was the leading aetiology in 109 (58.6%) patients, followed by viral causes in 43 (23.1%) and NAFLD in 19 (10.2%). Based on the MELD score, 94 (50.5%) had severe disease, 77 (41.4%) had moderate disease, and 15 (8.1%) had mild disease. Mean FT3 levels declined significantly from 3.21±0.20 in mild to 2.84±0.30 in moderate and 1.82±0.38 in severe cirrhosis ($p<0.0001$). Mean FT4 also decreased from 1.20±0.05 in mild to 1.15±0.05 in moderate and 1.02±0.07 in severe disease ($p<0.0001$). In contrast, the mean TSH level increased from 2.19±0.62 in mild to 3.26±0.99 in moderate and 5.05±1.04 in severe cirrhosis ($p<0.0001$). Low T3 syndrome was present in 104 patients (55.9%), predominantly in severe cases (87.5%), whereas normal thyroid status was more frequent in mild and moderate disease. Thyroid status was significantly associated with MELD severity ($p<0.0001$). **Conclusion:** Thyroid hormone alterations, particularly reduced FT3 levels, are associated with increasing MELD-based severity of liver cirrhosis. Thyroid profiles may serve as additional markers of hepatic dysfunction.

INTRODUCTION

Cirrhosis is the end stage of chronic liver disease, marked by diffuse fibrosis, regenerative nodules, and distortion of normal hepatic architecture.[1] Patients are classified as having compensated or decompensated cirrhosis. Decompensated cirrhosis presents with jaundice, ascites, hepatic encephalopathy, and variceal bleeding.[2] Compensated cirrhosis lacks these complications. This clinical distinction reflects differences in hepatic reserve and systemic metabolic function.[1,2]

The thyroid gland produces thyroxine (T4) and triiodothyronine (T3) hormones. These hormones regulate the basal metabolic rate, protein turnover, lipid metabolism, and carbohydrate utilisation.3 In circulation, most T3 and T4 are protein-bound, whereas free T3 (FT3) and free T4 (FT4) represent the biologically active fractions. Thyroid-stimulating hormone (TSH) from the anterior pituitary regulates their synthesis and release.[4]

The liver directly contributes to thyroid hormone metabolism. Hepatocytes participate in the peripheral conversion of T4 to T3 via deiodination. The liver also synthesises thyroid-binding globulin and other transport proteins that determine the total hormone concentration in the serum. Conjugation and biliary excretion of thyroid hormones occur in the liver. Progressive hepatocellular dysfunction disrupts these processes.5 Reduced binding protein synthesis and impaired peripheral conversion alter circulating hormone levels even in the absence of intrinsic thyroid disease.[6]

Patients with advanced cirrhosis often show reduced total T3 and FT3 levels. This reduction becomes more apparent with increasing hepatic dysfunction.7

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The decline in T3 levels reflects impaired hepatic conversion rather than primary hypothyroidism in many cases. FT4 and TSH values may remain within the reference range in the early stages but can change as the disease advances. These biochemical alterations parallel deterioration in hepatic synthetic capacity and metabolic regulation.[8]

Clinical assessment of cirrhosis severity increasingly relies on objective scoring systems. The Model for End-Stage Liver Disease (MELD) score was calculated using serum total bilirubin, serum creatinine, and international normalised ratio (INR). The MELD score estimates short-term mortality risk and stratifies patients across a spectrum of severity, independent of subjective clinical features.[9] Higher scores indicate advanced hepatic dysfunction and poorer prognosis.

Thyroid profile testing is not routinely included in the severity assessment of cirrhosis.10 Existing reports describe alterations in thyroid hormones in chronic liver disease, but a consistent association with MELD-based severity is not clearly defined in many hospital-based settings.

Data specifically examining FT3, FT4, and TSH in relation to the calculated MELD score after excluding primary thyroid disorders and major metabolic confounders remain limited. Clarifying this association can help determine whether thyroid hormone levels reflect graded hepatic dysfunction, as measured using an established prognostic scoring system. Therefore, this study aimed to evaluate thyroid dysfunction in patients with Liver Cirrhosis and its association with the severity of liver cirrhosis.

MATERIALS AND METHODS

This hospital-based observational study included 186 adult inpatients and outpatients with liver cirrhosis from the General Medicine Department at SRM Medical College Hospital and Research Centre, Trichy, over six months. Institutional Ethics Committee approval was obtained, and written informed consent was obtained from all patients.

Inclusion and exclusion criteria

Adults >18 years of age of either sex with a confirmed diagnosis of liver cirrhosis were included. Patients with pre-existing thyroid disorders in the absence of cirrhosis, malignancy, prior radiotherapy or chemotherapy, chronic kidney disease, nephrotic syndrome, or current use of levothyroxine, propylthiouracil, or carbimazole were excluded.

Methods

Patients were grouped based on the severity of liver cirrhosis using the MELD score. All enrolled participants underwent structured clinical assessments. A detailed medical history was obtained, and a complete physical examination was performed using a standard form. Liver cirrhosis was confirmed using biochemical liver function tests and ultrasonography of the abdomen and pelvis. After confirmation, venous blood samples were collected under aseptic precautions to estimate the thyroid profile, including FT3, FT4 and thyroid-stimulating hormone levels.

Serum creatinine, serum total bilirubin, and prothrombin time international normalised ratio were recorded for each patient. These parameters were used to calculate the MELD score using the revised formula: $9.57 \times \log_e(\text{creatinine}) + 3.78 \times \log_e(\text{total bilirubin}) + 11.2 \times \log_e(\text{INR}) + 6.43$. Patients were then categorised according to disease severity based on MELD scores. The relationship between thyroid hormone levels and liver disease severity was also examined.

Statistical Analysis

Statistical analyses were performed using IBM SPSS v25. Continuous variables were tested for normality and expressed as mean ± standard deviation. The mean FT3, FT4, and TSH levels across the three MELD severity categories (mild, moderate, and severe) were compared using one-way analysis of variance (ANOVA). Categorical variables are presented as frequencies and percentages. The association between thyroid status and MELD category was analysed using the chi-square test or Fisher's exact test, where appropriate. Statistical significance was set at $p < 0.05$.

RESULTS

Among the 186 patients, most were male (142, 76.3%). Alcohol was the predominant aetiology (109 patients, 58.6%). Based on the MELD score, severe, moderate, and mild disease were observed in 94 (50.5 %), 77 (41.4%), and 15 (8.1%) patients, respectively. Low T3 syndrome was present in 104 patients (55.9%), normal thyroid status in 79 (42.5%), and hypothyroidism in three (1.6%). [Table 1]

Table 1: Baseline characteristics and distribution of disease severity and thyroid status

Variable	Category	N (%)
Age group (years)	< 40	57 (30.6%)
	41–50	45 (24.2%)
	51–60	43 (23.1%)
	> 61	41 (22%)
Gender	Female	44 (23.7%)
	Male	142 (76.3%)
Aetiology	Alcohol	109 (58.6%)
	Cryptogenic	15 (8.1%)
	NAFLD	19 (10.2%)
	Viral	43 (23.1%)
MELD category	Mild	15 (8.1%)

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	Moderate	77 (41.4%)
	Severe	94 (50.5%)
Thyroid status	Hypothyroidism	3 (1.6%)
	Low T3 syndrome	104 (55.9%)
	Normal	79 (42.5%)

FT3 levels decreased across MELD categories, with mean values of 3.21 ± 0.20 in mild, 2.84 ± 0.30 in moderate, and 1.82 ± 0.38 in severe disease (P < 0.0001). FT4 also declined from 1.20 ± 0.05 in mild, to 1.15 ± 0.05 in moderate, and 1.02 ± 0.07 in severe cases (p < 0.0001). In contrast, TSH levels increased with severity, measuring 2.19 ± 0.62 in mild, 3.26 ± 0.99 in moderate, and 5.05 ± 1.04 in severe cirrhosis (p < 0.0001). [Table 2]

Table 2: Comparison of mean FT3, FT4, and TSH levels across MELD severity categories

Parameter	MELD Category			P value
	Mild	Moderate	Severe	
FT3	3.21 ± 0.20	2.84 ± 0.30	1.82 ± 0.38	<0.0001
FT4	1.20 ± 0.05	1.15 ± 0.05	1.02 ± 0.07	<0.0001
TSH	2.19 ± 0.62	3.26 ± 0.99	5.05 ± 1.04	<0.0001

Hypothyroidism was observed in two patients (66.7%) in the moderate category and one patient (33.3%) in the severe category, with no cases in the mild group. In low T3 syndrome, most patients belonged to the severe category 91 (87.5%). Normal thyroid status was observed in 15 patients (19%) with mild disease, 62 (78.5%) with moderate disease, and 2 (2.5%) with severe disease. The overall association between thyroid status and MELD category was significant (p < 0.0001). [Table 3]

Table 3: Distribution of thyroid status across MELD severity categories

Thyroid Status	MELD Category			P value
	Mild	Moderate	Severe	
Hypothyroidism	0	2 (66.7%)	1 (33.3%)	<0.0001
Low T3 syndrome	0	13 (12.5%)	91 (87.5%)	
Normal	15 (19%)	62 (78.5%)	2 (2.5%)	

DISCUSSION

This study shows that thyroid dysfunction is common in patients with liver cirrhosis and is associated with increasing disease severity. FT3 and FT4 levels decreased, while TSH levels increased with increasing MELD scores. Low T3 syndrome was the most common abnormality and was predominantly observed in patients with advanced liver disease. These findings indicate that changes in thyroid hormone levels are associated with the severity of hepatic dysfunction.

In our study, most participants were male, alcohol was the main cause, and severe liver disease predominated based on the MELD classification. Similarly, Badi et al. found that among 100 patients, males constituted 71 (71%) and females 29 (29%) of the patients. Alcohol was the leading cause of cirrhosis, identified in 66 patients (66%), followed by HBV in 12 (12%) and NASH in 11 (11%).[11] Kaur et al. reported that among 150 patients with cirrhosis, 128 (85.3%) were male and 22 (14.67%) were female, with a male-to-female ratio of 5.8:1. Alcoholic liver disease was the most common etiology, observed in 90 patients (60%), followed by NASH in 21 (14%), HCV in 16 (10.67%), and HBV in 5 (3.33%).[12]

Likewise, Punekar et al. reported that 100 patients with decompensated cirrhosis had ascites (74%), hepatic encephalopathy (38%), and coagulopathy (65%). Low T3 syndrome was present in 41% (41/100) of patients, hypothyroidism in 20%, and FT3 showed a significant negative correlation with MELD and Child-Pugh scores.[13] These studies show similar patient profiles with male predominance and alcohol as the main cause, along with advanced disease and reduced FT3 levels linked to higher severity scores, which directly supports our observed association between thyroid dysfunction and MELD severity.

In our study, FT3 and FT4 levels decreased with increasing liver disease severity, whereas TSH levels increased significantly. These findings indicate an association between hepatic dysfunction and changes in the thyroid hormone profile across MELD categories. Nabi and Rafiq found that patients with CLD had significantly lower FT3 and FT4 and higher TSH than controls (p < 0.001). FT3 levels were lower in patients with hepatic encephalopathy (n = 32) than in those without (n = 68). FT3 and FT4 were negatively correlated with MELD and CTP scores, whereas TSH was positively correlated with them.[14] Belu et al. reported a mean MELD score of 14.08 ± 4.32. MELD correlated positively with TSH at admission (ρ = 0.27, p < 0.001) and discharge (ρ = 0.21, p < 0.001), and negatively with T3 at discharge (ρ = −0.17, p < 0.001); fT4 showed no significant association.15 These studies show that as MELD scores rise, FT3 and FT4 fall while TSH increases, with significant associations to severity scores, directly supporting our finding that thyroid hormone changes reflect worsening liver function.

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Our study showed that low T3 syndrome was absent in mild disease and predominantly observed in severe cases, whereas normal thyroid status was more frequent in mild and moderate stages. Hypothyroidism was rare. Overall, thyroid status was significantly associated with MELD-based disease severity. Similarly, Hartl et al. reported that the median fT3 level declined with advanced stage (3.2 vs. 2.5 pg/ml; $p < 0.001$). Low fT3 levels occurred in 12.8% of patients, rising to 27.1% in S5 ($p = 0.015$). fT3 was inversely correlated with MELD ($\rho = -0.448$; $p < 0.001$) and predicted ACLF (28.9% vs. 5.8%) and liver-related death (28.9% vs. 3.5%; $p < 0.001$).[16] Belu et al. found that sick euthyroid syndrome (low FT3) was present in 29.6% (16/54) of patients. FT3 declined from 3.4±0.6 (Child A) to 2.7±0.5 (B) and 1.9±0.4 pg/ml (C) ($p<0.001$), with a negative correlation with MELD ($r=-0.48$, $p<0.01$) and Child-Pugh ($r=-0.52$, $p<0.01$).[17] Singh et al. showed that thyroid dysfunction increased with worsening Child-Pugh Class A 16.7% in Class A, 69.4% in Class B, and 90.0% in Class C. Across MELD risk groups, dysfunction was seen in 42.9%, 73.8%, 77.8%, and 100%, respectively.[18] These studies consistently show that FT3 levels fall and thyroid dysfunction becomes more common as MELD and Child-Pugh severity increase, confirming our observation that low T3 is closely linked to advanced liver disease.

Limitations

This single-centre study had a limited sample size. Its cross-sectional design prevents causal inferences. Thyroid function was assessed only once. Nutritional status, inflammation, and concurrent medications were not fully evaluated as potential confounding factors.

CONCLUSION

Thyroid hormone alterations are associated with the severity of liver cirrhosis. FT3 and FT4 levels decreased, whereas TSH levels increased with increasing MELD scores. Low T3 syndrome was more common in patients with advanced disease and was associated with greater liver dysfunction. These findings suggest that thyroid hormone changes may reflect the clinical severity of cirrhosis. Regular evaluation of thyroid function may help in assessing disease status. Further large-scale prospective studies are required to determine its role in prognosis and clinical management.

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