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Original Research Article

THYROID FUNCTION ABNORMALITIES AND THEIR ASSOCIATION WITH CHILD–PUGH SEVERITY IN PATIENTS WITH LIVER CIRRHOSIS: A CROSS-SECTIONAL STUDY

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ABSTRACT

Background: Liver cirrhosis may alter thyroid hormone conversion, transport, and clearance, resulting in abnormal thyroid function tests even in the absence of primary thyroid disease. The objective is to evaluate abnormalities in serum free triiodothyronine (fT3), free thyroxine (fT4), and thyroid-stimulating hormone (TSH) levels and determine their association with liver cirrhosis severity according to the Child–Pugh classification.

Materials and Methods: This hospital-based observational cross-sectional study included 100 adult patients with liver cirrhosis admitted to the Department of General Medicine, GMERS Medical College and Hospital, Himmatnagar, Gujarat, between July 2024 and June 2025. Demographic, clinical, hematological, biochemical, coagulation, and ultrasonographic findings were recorded. Liver disease severity was assessed using the Child–Pugh score. Serum fT3, fT4, and TSH were measured by electrochemiluminescence immunoassay. Continuous variables were expressed as mean ± standard deviation and categorical variables as frequency and percentage. Thyroid parameters were compared between Child–Pugh Classes B and C using the independent-samples t-test, with $p<0.05$ considered statistically significant.

Results: The mean age was 56.64 ± 10.65 years, and all participants were male. Child–Pugh Class B and Class C accounted for 44% and 56% of patients, respectively. Mean fT3, fT4, and TSH levels were 3.43 ± 0.76 pg/mL, 1.38 ± 0.40 ng/dL, and 3.44 ± 2.97 µIU/mL, respectively. TSH ≥5 µIU/mL was observed in 13% of patients. No significant differences were found between Classes B and C for fT3 ($p=0.396$), fT4 ($p=0.332$), or TSH ($p=0.486$).

Conclusion: Thyroid function abnormalities were observed in advanced liver cirrhosis; however, fT3, fT4, and TSH levels were not significantly associated with Child–Pugh severity in the present cohort.

Keywords: Liver cirrhosis, thyroid function, free T3, free T4, TSH, Child–Pugh score.

INTRODUCTION

Liver cirrhosis is the end stage of chronic hepatic injury, characterized by fibrosis, regenerative nodules, and progressive loss of liver architecture, resulting in impaired synthetic and detoxification functions. It may remain compensated initially but progresses to decompensation with ascites, variceal bleeding, jaundice, and hepatic encephalopathy,

followed by complications such as spontaneous bacterial peritonitis and hepatorenal syndrome.[1]

The liver regulates thyroid hormone metabolism, transport, and clearance. T3 and T4 circulate mainly bound to thyroxine-binding globulin, transthyretin, and albumin, and exert their metabolic effects through nuclear thyroid hormone receptors.[2] Hepatic tissue is central to extra-thyroidal thyroid hormone metabolism, with type 1 iodothyronine deiodinase converting T4 to biologically active T3 and contributing approximately 30–40% of circulating T3. In liver cirrhosis, particularly during decompensation, reduced deiodinase activity leads to decreased fT3 and increased reverse T3, producing the characteristic pattern of non-thyroidal illness syndrome or euthyroid sick syndrome despite the absence of intrinsic thyroid disease.[3,4]

The liver synthesizes thyroid hormone–binding proteins, particularly TBG and albumin, and contributes to the clearance of TSH and thyroid hormone conjugates through biliary excretion; consequently, hepatic dysfunction can alter circulating thyroid hormone concentrations.[5] Conversely, thyroid hormones regulate key hepatic processes, including mitochondrial oxidative phosphorylation, bile acid synthesis, and cytochrome P450 activity. This bidirectional relationship explains why hyperthyroidism may be associated with hepatic steatosis and elevated transaminases, whereas hypothyroidism may contribute to cholestasis and progression of non-alcoholic fatty liver disease.[6]

The Child–Pugh score assesses cirrhosis severity using serum bilirubin, albumin, INR, ascites, and encephalopathy, and classifies patients into Classes A, B, and C. However, it does not capture endocrine or metabolic disturbances; therefore, fT3 and TSH have been proposed as adjunctive markers of hepatic reserve and worsening liver dysfunction.[7] Existing literature has identified a trend toward lower fT3 and higher TSH concentrations in patients with more advanced stages of cirrhosis, suggesting that these hormonal shifts are not merely epiphenomena but may reflect the severity of hepatic compromise.[8]

Given the close interplay between hepatic and thyroid function, evaluating fT3, fT4, and TSH in cirrhosis may provide additional insight into disease severity.[9] Therefore, the present study was undertaken to evaluate abnormalities in serum fT3, fT4, and TSH levels and to determine their association with the severity of liver cirrhosis according to the Child–Pugh classification.

MATERIALS AND METHODS

Study Setting, Duration and study design Type: A hospital-based observational cross-sectional study was conducted in the Department of General Medicine at GMERS Medical College and Hospital, Himmatnagar, Gujarat, over a one-year period from July 2024 to June 2025.

Study Participants, Inclusion and Exclusion Criteria

Adult patients aged more than 18 years and up to 80 years, of either sex, with known or newly diagnosed liver cirrhosis established on the basis of clinical history, biochemical findings and abdominal ultrasonography were eligible for inclusion after providing written informed consent. Patients with a pre-existing thyroid disorder unrelated to cirrhosis, multiorgan failure, malignancy, ongoing radiotherapy or chemotherapy, active infection, chronic renal disease including nephrotic syndrome, or cardiac, pancreatic or musculoskeletal disease were excluded. Patients receiving medications known to interfere with thyroid metabolism, including levothyroxine, propylthiouracil, carbimazole, iodine, amiodarone and beta-blockers, were also excluded.

Sample Size and Sampling

The sample size was calculated using an anticipated prevalence of hypothyroidism of 62%, (Patira Nk et al.[9]) a 95% confidence level and an absolute precision of 10%. The minimum required sample size was 91; however, 100 eligible patients were included in the final analysis. All eligible patients presenting during the study period were screened consecutively, although the thesis described the sampling technique as purposive sampling.

Study Tools, Definitions and Criteria

Data were recorded using a predesigned proforma that included demographic characteristics, alcohol and smoking history, etiology and duration of cirrhosis, clinical complications and relevant general and systemic examination findings. Laboratory investigations included complete blood count, liver and renal function tests, serum electrolytes, prothrombin time, international normalized ratio, viral markers and abdominal ultrasonography. Cirrhosis severity was assessed using the Child–Pugh score, comprising serum bilirubin, serum albumin, INR, ascites and hepatic encephalopathy, and patients were classified into Child–Pugh Classes A, B or C. Serum free triiodothyronine, free thyroxine and thyroid-stimulating hormone were measured in fasting samples using electrochemiluminescence immunoassay. The laboratory reference ranges were 2.1–4.4 pg/mL for free T3, 0.8–2.7 ng/dL for free T4 and 0.35–5.5 µIU/mL for TSH. For the study-defined prevalence analysis, a TSH value of at least 5 µIU/mL was used as the threshold.

Data Collection

The study was approved by the institutional ethics committee. Eligible participants received a participant information sheet in their native language and were informed about the study objectives, procedures, potential benefits and risks. Written informed consent was obtained before enrolment, and participation involved no additional financial burden or compromise in clinical care.

Confidentiality and privacy were maintained throughout the study. Demographic details, clinical history and examination findings were recorded at admission, followed by laboratory investigations, ultrasonographic evaluation, Child–Pugh assessment and fasting thyroid-function testing.

Data Analysis

Data were entered and analysed using Epi Info CDC version 7. Continuous variables were summarized as mean and standard deviation, whereas categorical variables were expressed as frequencies and percentages. Associations between categorical variables were assessed using the chi-square test, while continuous variables were compared using the independent-samples t-test. A p-value below 0.05 was considered statistically significant.

RESULTS

As shown in [Table 1], the cohort had a mean age of 56.64 ± 10.65 years, all participants were male, alcohol consumption was present in all patients and 32% reported smoking. Abdominal distension was universal, while pedal edema and jaundice-related symptoms were present in approximately half of the participants.

Table 1: Demographic and Clinical Characteristics of the Study Participants (n=100)

Characteristic	Value
Demographic and exposure characteristics	
Age, years	56.64 ± 10.65
Age range, years	35–74
Male sex	100 (100.0)
Alcohol consumption	100 (100.0)
Smoking	32 (32.0)
Presenting clinical features	
Abdominal distension	100 (100.0)
Pedal edema	50 (50.0)
Yellow urine	46 (46.0)
Yellow sclera	46 (46.0)
Breathlessness	16 (16.0)
General examination findings	
Pallor	60 (60.0)
Icterus	46 (46.0)
Clubbing	40 (40.0)
Dehydration	32 (32.0)

Data are presented as mean ± standard deviation, range or number (percentage), as appropriate.

[Table 2] demonstrated marked hematological and hepatic derangements, including a mean hemoglobin level of 7.61 ± 0.95 g/dL, platelet count of 136,554 ± 66,488/mm³, total bilirubin of 4.34 ± 4.03 mg/dL, albumin of 2.31 ± 0.42 g/dL, and INR of 1.49 ± 0.47; renal and electrolyte parameters were relatively preserved, whereas ultrasonography revealed surface nodularity, coarse echotexture, ascites, and portal-vein dilatation in all patients, with fatty liver changes in 52%, and 56% of participants were categorized as Child–Pugh Class C.

Table 2: Hematological, Biochemical, Ultrasonographic and Liver-Disease Severity Characteristics (n=100)

Parameter	Value
Hematological parameters	
Hemoglobin, g/dL	7.61 ± 0.95
Total leukocyte count, cells/mm³	8,012 ± 2,275
Platelet count, cells/mm³	136,554 ± 66,488
Liver-function parameters	
Total bilirubin, mg/dL	4.34 ± 4.03
Direct bilirubin, mg/dL	2.83 ± 2.82
Sspartate aminotransferase, U/L	94.80 ± 52.40
alanine aminotransferase, U/L	99.74 ± 52.93
Serum albumin, g/dL	2.31 ± 0.42
Coagulation parameters	
Prothrombin time, seconds	17.84 ± 5.59
International normalized ratio	1.49 ± 0.47
Renal function and electrolytes	
Blood urea, mg/dL	31.78 ± 11.30
Serum creatinine, mg/dL	0.98 ± 0.33
Serum sodium, mEq/L	137.56 ± 4.92
Serum potassium, mEq/L	4.26 ± 0.45
Ultrasonographic findings	
Surface nodularity of liver	100 (100.0)
Coarse heterogeneous echotexture	100 (100.0)
Ascites	100 (100.0)
Portal-vein diameter >13 mm	100 (100.0)

Fatty liver changes	52 (52.0)
Child–Pugh classification	
Class B	44 (44.0)
Class C	56 (56.0)

Data are presented as mean ± standard deviation or number (percentage).

[Table 3] revealed mean free T3, free T4, and TSH levels of 3.43 ± 0.76 pg/mL, 1.38 ± 0.40 ng/dL, and 3.44 ± 2.97 µIU/mL, respectively, with 13% of patients having TSH values ≥5 µIU/mL.

Table 3: Thyroid Function Profile and Distribution According to the Study-Defined TSH Threshold (n=100)

Thyroid parameter	Value
Free T3, pg/mL	3.43 ± 0.76
Free T4, ng/dL	1.38 ± 0.40
TSH, µIU/mL	3.44 ± 2.97
TSH ≥5 µIU/mL	13 (13.0)
TSH <5 µIU/mL	87 (87.0)

Table 4: Comparison of Thyroid Function Parameters Between Child–Pugh Classes B and C

Thyroid parameter	Child–Pugh Class B (n=44)	Child–Pugh Class C (n=56)	Mean difference*	95% CI	p-value
Free T3, pg/mL	3.36 ± 0.90	3.49 ± 0.63	−0.13	−0.43 to 0.17	0.396
Free T4, ng/dL	1.34 ± 0.33	1.41 ± 0.44	−0.08	−0.24 to 0.08	0.332
TSH, µIU/mL	3.68 ± 3.07	3.26 ± 2.91	0.42	−0.77 to 1.61	0.486

Mean difference was calculated as Child–Pugh Class B minus Child–Pugh Class C. Comparisons were performed using the independent-samples t-test.

[Table 4] found no statistically significant differences in free T3, free T4, or TSH between Child–Pugh Classes B and C, with corresponding p-values of 0.396, 0.332, and 0.486, indicating that thyroid hormone levels did not vary significantly with Child–Pugh severity in the present cohort.

DISCUSSION

Liver cirrhosis affects not only hepatic architecture and function but also several endocrine pathways, particularly thyroid hormone conversion, transport, and clearance. These alterations may produce abnormal thyroid function tests even in the absence of primary thyroid disease, making their interpretation clinically relevant in patients with advanced cirrhosis.

In the present study, the mean age was 56.64 ± 10.65 years, and all participants were male. In comparison, Sangole et al,[10] reported a lower mean age of 46.93 ± 10.94 years with 81% male participants, while Prasad et al,[11] also observed predominance of middle-aged men. Alcohol consumption was documented in all patients in the present study, and 32% also reported smoking. Clinically, abdominal distension was present in all patients, pedal edema in 50%, and jaundice-related manifestations in 46%, indicating that the study population largely represented decompensated cirrhosis.

The present study demonstrated marked hematological and biochemical abnormalities. Mean hemoglobin was 7.61 ± 0.95 g/dL and mean platelet count was 136,554 ± 66,488/mm³, indicating anemia and thrombocytopenia. Sangole et al,[10] similarly reported a median hemoglobin of 8.1 g/dL and platelet count of 91,000/mm³. Mean total bilirubin was 4.34 ± 4.03 mg/dL, albumin was 2.31 ± 0.42 g/dL, and INR was 1.49 ± 0.47 in the present study, reflecting impaired hepatic excretory and synthetic function. Comparable abnormalities were reported by Prasad et al,[11] and Patira et al,[9] Renal function and electrolyte levels remained relatively preserved in the present cohort.

In the present study, ultrasonography revealed surface nodularity, coarse heterogeneous echotexture, ascites, and portal-vein diameter >13 mm in all patients, while fatty liver changes were present in 52%. In comparison, Punekar et al,[12] reported ascites in 74% and portal-vein dilatation in 68%, whereas Chaudhary et al.13 reported coarse echotexture in 82%, ascites in 71%, and portal-vein dilatation in 65%. The higher frequency of these findings in the present study suggests inclusion of a more uniformly advanced cirrhotic population. Child–Pugh Class B and Class C constituted 44% and 56% of patients, respectively, closely resembling the 42% and 58% distribution reported by Prasad et al.[11]

The mean fT3, fT4, and TSH levels in the present study were 3.43 ± 0.76 pg/mL, 1.38 ± 0.40 ng/dL, and 3.44 ± 2.97 µIU/mL, respectively. Sangole et al.10 reported lower mean fT3 and fT4 levels of 2.34 ± 0.79 pg/mL and 1.21 ± 0.36 ng/dL, while mean TSH was comparable at 3.12 ± 1.84 µIU/mL. Prasad et al,[11] also observed lower fT3 levels of approximately 2.3 pg/mL. Thus, fT3 appeared relatively preserved in the present study, possibly because patients with active infection and multiorgan failure were excluded. TSH ≥5 µIU/mL was observed in 13% of patients in the present study, compared with 18% reported by Prasad et al,[11] and 19.1% by Sangole et al,[10] Differences in

laboratory thresholds, disease severity, assay methods, and patient selection may explain this variation.

The principal finding of the present study was that thyroid function parameters did not differ significantly between Child–Pugh Classes B and C. Mean fT3 was 3.36 ± 0.90 pg/mL in Class B and 3.49 ± 0.63 pg/mL in Class C, while mean fT4 was 1.34 ± 0.33 and 1.41 ± 0.44 ng/dL, respectively. Mean TSH was 3.68 ± 3.07 µIU/mL in Class B and 3.26 ± 2.91 µIU/mL in Class C. The corresponding p-values were 0.396, 0.332, and 0.486. In contrast, Punekar et al,[12] and Waqas et al,[14] reported a progressive decline in fT3 with worsening Child–Pugh class. The absence of such a trend in the present study may be related to the lack of Child–Pugh Class A patients, limited biological separation between Classes B and C, and exclusion of patients with severe systemic illness.

The present study was limited by its single-centre cross-sectional design, modest sample size, exclusively male population, absence of a control group, and lack of Child–Pugh Class A patients. Nevertheless, the findings indicate that elevated TSH values may occur in advanced cirrhosis, while fT3, fT4, and TSH may not reliably differentiate between Child–Pugh Classes B and C.

CONCLUSION

The present study found that thyroid function abnormalities were present among patients with advanced liver cirrhosis, with 13% showing TSH levels ≥5 µIU/mL; however, mean fT3, fT4, and TSH levels did not differ significantly between Child–Pugh Classes B and C. These findings suggest that thyroid function test abnormalities may accompany cirrhosis but may not reliably reflect disease severity within patients who already have moderate-to-severe hepatic dysfunction. Therefore, thyroid parameters should be interpreted in conjunction with clinical status and liver disease severity rather than as isolated markers of progression.

REFERENCES

1. Battle A, Mudd J, Ahlenstiel G, Kalo E. Liver cirrhosis: evolving definitions, and recent advances in diagnosis, prevention and management. Livers. 2025;5(3):28.
2. Chng CL, Goh GBB, Yen PM. Metabolic and functional cross talk between the thyroid and liver. Thyroid. 2025;35:thy.2025.0178.
3. Soares de Oliveira L, Ritter MJ. Thyroid hormone and the liver. Hepatol Commun. 2025;9(1):e0000596.
4. Ritter MJ, Amano I, Hollenberg AN. Thyroid hormone signaling and the liver. Hepatology. 2020;72(2):742–52.
5. Huang YY. Cross-talk between the thyroid and liver: a new target for nonalcoholic fatty liver disease treatment. World J Gastroenterol. 2013;19(45):8238–46.
6. Sinha RA, Singh BK, Yen PM. Direct effects of thyroid hormones on hepatic lipid metabolism. Nat Rev Endocrinol. 2018;14(5):259–69.
7. Gülcicegi DE, Goeser T, Kasper P. Prognostic assessment of liver cirrhosis and its complications: current concepts and future perspectives. Front Med. 2023;10:1268102.
8. Hartl L, Simbrunner B, Jachs M, Wolf P, Bauer DJM, Scheiner B, et al. Lower free triiodothyronine levels in cirrhosis are linked to systemic inflammation, acute-on-chronic liver failure, and mortality. JHEP Rep. 2024;6(1):100954.
9. Patira DNK. Correlation of thyroid function tests with severity of liver dysfunction. J Med Sci Clin Res. 2017;5(5):21921–7.
10. Sangole SP, Salagre K. Thyroid profile in liver cirrhosis patients. J Assoc Physicians India. 2024;72(9):22–5.
11. Prasad D, Singh R, Sahu S, Pathak A. Evaluation of thyroid function test in chronic liver disease. Int J Pharm Clin Res. 2024;16(6):1404–10.
12. Punekar P, Sharma A, Jain A. Thyroid dysfunction in liver cirrhosis. Indian J Endocrinol Metab. 2018;22(5):645–9.
13. Chaudary S, Shahi A, Jaiswal N, Dhakal P, Khatri P, Pandey S, et al. Thyroid function abnormalities in liver cirrhosis. J Diabetes Endocrinol Assoc Nepal. 2019;3(2):25–31.
14. Waqas, Uthman M. Effect of liver cirrhosis on thyroid hormone levels. Pak J Med Health Sci. 2021;15(4):768–72.