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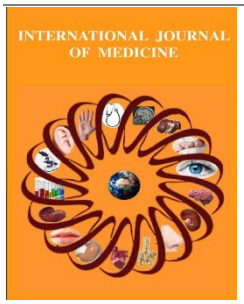

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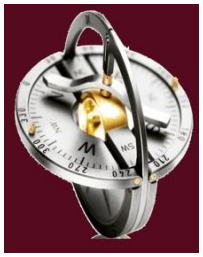

Review Article

To Study the Correlation of Thyroid Hormone Deficiency in Cirrhotic Patients in a Tertiary Care Hospital: A Case Control Study

Tanya.[1], Tarundeep[2], Sunil Kumar Sharma[3*], Kusum Bali[4], Sunil Singh Bains[5], Aparna Singh[6]

1Department of Medicine, MBBS Intern, Punjab Institute of Medical Sciences Jalandhar, Punjab, India
2Department of Medicine, Associate Professor, Punjab Institute of Medical Sciences Jalandhar, Punjab, India
3Department of Medicine, Assistant Professor, Punjab Institute of Medical Sciences Jalandhar, Punjab, India
4Department of Medicine, Professor, Punjab Institute of Medical Sciences Jalandhar, Punjab, India
5Department of Medicine, MBBS Intern, Punjab Institute of Medical Sciences Jalandhar, Punjab, India
6Department of Medicine, MBBS Intern, Punjab Institute of Medical Sciences Jalandhar, Punjab, India.

Abstract

Background: In India liver disorders are becoming a notable public health issue, encouraging early screening to detect and assess the disease severity. Liver cirrhosis affects the metabolism of thyroid hormones which include fT3, fT4 and TSH. This study aims at evaluating the correlation of thyroid profile with cirrhosis using Child Pugh score (CTP), model for end-stage liver disease (MELD), MELD 3.0 and MELD-sodium. ***Objectives***: To assess the thyroid hormone deficiency in cirrhotic patients and compare it with the normal population. Additionally, evaluating the correlation between thyroid hormone deficiency and severity of liver cirrhosis. ***Material and methods:*** Our case-control study included 70 cases and 70 controls both age and gender-matched. The study population is obtained from our tertiary care center. The results of thyroid profile were correlated using CTP, MELD, MELD 3.0 and MELD sodium scoring systems. ***Results***: Cirrhotic patients showed markedly reduced fT3 and fT4 levels with significantly elevated TSH (all $p < 0.01$) compared to controls, indicating thyroid dysfunction associated with hepatic impairment. Clinical hypothyroidism was observed in 78.6% and subclinical in 21.4% of cases, showing a significant association with higher CTP grades ($p = 0.026$). Patients with clinical hypothyroidism also had higher MELD 3.0, MELD, and MELD-Na scores ($p < 0.05$), suggesting that worsening thyroid function parallels increasing liver disease severity.

Keywords: Cirrhotic liver, MELD 3.0, MELD sodium, a case-control study, thyroid hormone deficiency

Introduction:

Liver cirrhosis is a preeminent cause of morbidity and mortality globally, with its prevalence rising by 74.53% between 1990 and 2017. Cirrhosis is characterized by the development of fibrosis and nodules in the liver as a result of chronic damage, leading to a disarrangement of the liver's normal lobular structure [1]. It is classified clinically as "compensated" or "decompensated"[2]. The liver has a crucial role in producing carrier proteins and metabolizing different hormones [3]. The liver performs a vital role in the metabolism of thyroid hormones, serving as the primary organ for the peripheral conversion of tetraiodothyronine (T4) into triiodothyronine (T3) through the action of Type 1 deiodinase. Both hormones are attached to plasma proteins, including thyroxine-binding globulin (a glycoprotein produced in the liver with a plasma half-life of 5 days), transthyretin (previously known as thyroxine-binding prealbumin), and albumin, which has a low affinity but high binding capacity for thyroid hormones, accounting for the remaining 10%–20% of serum T3 and T4 [4,5]. Liver failure leads to elevated levels of circulating endotoxins and pro-inflammatory mediators, resembling the clinical condition of sepsis, which in turn causes dysfunction in endocrine glands. This condition is often referred to as sick euthyroid syndrome or nonthyroidal illness syndrome (NTIS) [6]. Decreased levels of fT3 and fT4 is found in 72.5 % and 26.4% of cirrhosis patients respectively whereas TSH is found to be increased in about 52.3 % population suffering from cirrhosis as per a study conducted on 102 patients in 2017 [7], showing that the prevalence of thyroid profile derangement exists in 13 to 61 %. T4 and T3 execute a important role in regulating the basal metabolic rate of all cells, including liver cells (hepatocytes), and thus influence liver function [8]. A review of the literature indicates that there is insufficient research material on thyroid hormone deficiency and its correlation with severity of liver cirrhosis. In this study we will compare cases of cirrhosis with controls having no comorbidities in view of thyroid hormone deficiency. This study aims to explore the correlation between thyroid hormone deficiency and cirrhosis, with the purpose of using it as a prognostic index, thereby permitting timely interventions..

Address for Correspondence: Sunil Kumar Sharma, Department of Medicine, Assistant Professor, Punjab Institute of Medical Sciences Jalandhar, Punjab, India. sunilkumarsharma496@gmail.com

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Methodology

This is a prospective case-control study performed at our tertiary care centre in Punjab Institute of Medical Sciences, Jalandhar, Punjab, India for 6 months after acquiring approval from the Ethics Committee of the Institution. A total of 140 patients were included in the study. 70 consenting patients aged >18-80 years males and females diagnosed with cirrhosis, admitted to a tertiary care hospital were included in the study as cases. For controls, patients aged >18-80 years male and female who presented to medicine OPD for routine check-up and didn't have any comorbidities like diabetes, hypertension, hypothyroidism, cirrhosis liver and chronic kidney disease etc, were included in the study after obtaining the consent. The study lasted from June 2025 to November 2025. Exclusion criteria included (a) Patients with previously diagnosed thyroid disorders without cirrhosis; (b) Patients with previous exposure to radiotherapy, chemotherapy and thyroid surgery; (c) Patients with malignancy; (d) Patients with severe multiorgan failure; (e) Patients who are taking drugs interfering with thyroid metabolism (levothyroxine, methimazole, propylthiouracil, iodine, amiodarone); (h) Pregnant and breastfeeding patients; (i) Patients who refused to give consent.

After applying the necessary criteria, sample size was calculated which is 70 cases and 70 controls. The diagnosis of cirrhosis was made both clinically and radiologically via symptoms of liver failure including fatigue, loss of hair, itchy skin, weight loss, decreased appetite, jaundice, ascites, gastrointestinal bleeding, confusion, drowsiness and hepatic encephalopathy, edema, signs such as pallor, icterus, enlargement of parotid gland, spider telangiectasias, palmer erythema, gynaecomastia, atrophy of testis, thyroid gland examination, blood tests covering complete blood count, liver function tests, renal function tests, PTI/INR, viral makers, fT3, fT4, thyroid stimulating hormone (TSH) and ultrasound of whole abdomen. Thyroid profile of each patient was obtained. CTP, MELD, MELD 3.0 and MELD sodium scores were calculated.

Statistical analysis: Data so collected was tabulated in an excel sheet, under the guidance of statistician. The means and standard deviations of the measurements per group were used for statistical analysis (SPSS 22.00 for windows; SPSS inc, Chicago, USA). For each assessment point, data were statistically analyzed using t test and chi square test. The level of significance was set at $p < 0.05$.

RESULTS

Table 1: Baseline data among the study groups

Variables	Case		Control		p value
	N=70	%	N=70	%	
Gender					
Male	59	84.3	47	67.1	0.10
Female	11	15.7	23	32.9	
Age in years (Mean±SD)	50.19±12.81		44.37±13.22		0.16

*: statistically significant

The study included 70 cases with cirrhosis and 70 control subjects. The gender distribution showed that 59 of the cases were male, compared to 47 in the control group, with a p-value of 0.10, indicating no statistically significant difference. The female proportion was 11 among cases and 23 among controls. The mean age of the cirrhosis group was 50.19 ± 12.81 years, while the control group had a mean age of 44.37 ± 13.22 years. These data suggest that the two groups were comparable in terms of gender and age, providing a suitable baseline for evaluating the effect of cirrhosis on thyroid profile.

Table 2: Comparison of investigative profile among the study groups

Group		Urea	Creatinine	Sodium	Potassium	Albumin	AST	ALT	ALP	PTI/INR	HB	TLC	PLT
Case	Mean	64.93	2.31	137.43	4.29	2.77	152.47	133.6	184.53	1.74	10.17	10467.59	1.61
	SD	52.49	2.29	7.19	.93	1.01	309.27	368.6	149.06	.58	2.48	5884.57	.94
Control	Mean	33.66	.98	140.73	5.23	4.03	43.90	60.0	109.31	4.34	12.98	8164.52	2.31
	SD	11.86	.26	3.84	4.99	.81	23.19	33.71	36.23	1.15	1.44	2119.06	.55
p value		<0.01*	<0.01*	0.001*	0.12	<0.01*	0.004*	0.098	<0.01*	<0.01*	<0.01*	0.002*	<0.01*

*: statistically significant

A comparison of the biochemical and hematological parameters between the case and control groups revealed significant differences in several variables. The mean serum urea and creatinine levels were markedly higher in cirrhotic patients (64.93 ± 52.49 mg/dL and 2.31 ± 2.29 mg/dL, respectively) compared to controls (33.66 ± 11.86 mg/dL and 0.98 ± 0.26 mg/dL, both $p < 0.01$), indicating impaired renal function among cases. The mean serum sodium level was significantly lower in cases (137.43 ± 7.19 mmol/L) than in controls (140.73 ± 3.84 mmol/L, $p = 0.001$), while the difference in serum potassium was not statistically significant ($p = 0.12$). The mean serum albumin concentration was substantially reduced in cirrhotic patients (2.77 ± 1.01 g/dL) compared to controls (4.03 ± 0.81 g/dL, $p < 0.01$), reflecting compromised hepatic synthetic function.

The liver enzyme profile also showed derangements, with AST and ALP levels being significantly higher in the case group ($p = 0.004$ and $p < 0.01$, respectively), while the difference in ALT was not statistically significant ($p = 0.098$). Coagulation parameters, represented by PT/INR, were markedly prolonged among cirrhotic patients (1.74 ± 0.58) compared to controls (4.34 ± 1.15, $p < 0.01$). Hematological indices showed a significant reduction in hemoglobin levels (10.17 ± 2.48 g/dL vs. 12.98 ± 1.44 g/dL, $p < 0.01$), along with lower platelet counts ($1.61 \pm 0.94 \times 10^5$/μL vs. $2.31 \pm 0.55 \times 10^5$/μL, $p < 0.01$). The total leukocyte count was also significantly different between the two groups ($p = 0.002$).

These findings collectively indicate that cirrhotic patients exhibited significant hepatic, renal, and hematological abnormalities, consistent with the multisystem impact of advanced liver disease and supporting the observed association between thyroid hormone deficiency and cirrhosis severity.

Table 3: Distribution of CTP and MELD score among the cases

Variables	N=70	%
CTP Score		
A	1	1.4
B	26	37.2

C	43	61.4
MELD 3.0 (Mean±SD)	26.66±6.22	
MELD (Mean±SD)	24.59±6.34	
MELD Na (Mean±SD)	25.26±6.35	

The distribution of patients according to the Child-Turcotte-Pugh (CTP) score showed that 1.4% of the patients were classified as CTP class A, indicating well-compensated liver function. The majority of patients, 37.2%, fell into class B, while 61.4% were categorized as class C, reflecting more severe liver disease.

The study assessed liver disease severity using three different MELD scoring systems: MELD 3.0, MELD, and MELD Na (which includes serum sodium). The overall mean MELD score was 24.59 ± 6.34. Specifically, the mean MELD 3.0 score was higher at 26.66 ± 6.22, indicating a more refined assessment without sodium. The MELD Na score, which accounts for serum sodium levels, had a mean of 25.26 ± 6.35. These scores collectively reflect the varying degrees of liver dysfunction among the patients and are useful for analyzing their relationship with thyroid profile alterations in this population.

Table 4: Comparison of thyroid function parameters between cases and controls

Group		fT3	fT4	TSH
Case	Mean	1.982	9.504	10.547
	SD	.585	2.727	4.708
Control	Mean	4.321	16.605	2.672
	SD	1.056	3.056	1.118
t test		11.78	19.132	37.191
p value		<0.01*	<0.01*	<0.01*

*: statistically significant

In the case group, the mean levels of fT3, fT4, and TSH were 1.982, 9.504, and 10.547, respectively, with standard deviations of 0.585, 2.727, and 4.708. In contrast, the control group exhibited higher mean values for fT3 (4.321), fT4 (16.605), and lower mean TSH (2.672), with standard deviations of 1.056, 3.056, and 1.118. The t-test results show significant differences between the groups for all parameters, with t-values of 11.78 for fT3, 19.132 for fT4, and 37.191 for TSH. The corresponding p-values were all less than 0.01, indicating highly significant differences in thyroid function parameters between the cases and controls.

Table 5: Type of hypothyroidism according to CTP grade

			Thyroidism Type		
			Clinical	Subclinical	Total
CTP SCORE	A	N	1	0	1
		%	1.8%	0.0%	1.4%
	B	N	15	11	26
		%	27.3%	73.3%	37.1%
	C	N	39	4	43
		%	70.9%	26.7%	61.4%
Total		N	55	15	70
		%	100.0%	100.0%	100.0%
Chi Square			11.89		
p value			0.026*		

*: statistically significant

A significant association was observed between the Child–Turcotte–Pugh (CTP) score and the type of thyroid dysfunction among the study population. Among patients with CTP class A, 1.8% had clinical hypothyroidism, while none had subclinical hypothyroidism. In CTP class B, 27.3% of patients had clinical hypothyroidism and 73.3% had subclinical hypothyroidism. In contrast, CTP class C was associated predominantly with clinical hypothyroidism (70.9%) compared to subclinical hypothyroidism (26.7%). Overall, out of 70 patients, 55 (78.6%) had clinical hypothyroidism and 15 (21.4%) had subclinical hypothyroidism. The difference in distribution of thyroid dysfunction across CTP classes was statistically significant (χ^2 = 11.89, p = 0.026), indicating a trend toward more severe thyroid dysfunction with worsening liver disease severity.

Table 6: Mean MELD score according to type of hypothyroidism

Thyroidism Type		MELD 3.0 SCORE	MELD	MELD NA
Clinical	Mean	28.31	25.16	25.80
	SD	4.26	3.46	4.55
Subclinical	Mean	24.27	22.47	23.27
	SD	4.62	3.54	3.28
t test		3.90	3.17	1.90
p value		0.0031*	0.038*	0.046*

The relationship between thyroid dysfunction and liver disease severity, as assessed by MELD-based scores, was also analyzed. Patients with clinical hypothyroidism had significantly higher mean scores compared to those with subclinical hypothyroidism. The mean MELD 3.0 score was 28.31 ± 4.26 in the clinical group and 24.27 ± 4.62 in the subclinical group (t = 3.90, p = 0.0031). Similarly, the mean MELD score was 25.16 ± 3.46 for clinical hypothyroidism and 22.47 ± 3.54 for subclinical hypothyroidism (t = 3.17, p = 0.038). The MELD-Na score also showed a comparable trend, with values of 25.80 ± 4.55 in clinical hypothyroidism versus 23.27 ± 3.28 in subclinical hypothyroidism (t = 1.90, p = 0.046). These findings indicate that patients with clinical hypothyroidism tend to have more severe liver dysfunction as reflected by significantly higher MELD-based scores

DISCUSSION

The current study evaluated 70 patients with liver cirrhosis and 70 healthy controls. Among the cirrhotic group, 84.3% were males and 15.7% were females, while in the control group, 67.1% were males

and 32.9% were females. The mean age of patients with cirrhosis was 50.19 ± 12.81 years, compared to 44.37 ± 13.22 years in controls, and this difference was not statistically significant (p = 0.16). These results indicate that both groups were comparable in age and gender distribution. A similar demographic pattern was observed by Punekar et al. (2018), who found a male predominance (71%) among cirrhotic patients, with comparable mean ages between the case (43 ± 14 years) and control (42 ± 15 years) groups, all of whom had decompensated liver cirrhosis.

In the present study, cirrhotic patients showed significantly higher mean urea (64.93 ± 52.49 mg/dL) and creatinine (2.31 ± 2.29 mg/dL) levels compared with controls (33.66 ± 11.86 mg/dL and 0.98 ± 0.26 mg/dL, respectively; $p < 0.01$), while serum albumin was notably reduced (2.77 ± 1.01 g/dL vs. 4.03 ± 0.81 g/dL; $p < 0.01$). Liver enzymes (AST and ALP) and PT/INR were significantly elevated among cases, indicating hepatic dysfunction. Thyroid hormone analysis revealed lower fT3 (1.98 ± 0.58 pg/mL vs. 4.32 ± 1.05 pg/mL) and fT4 (9.50 ± 2.72 pmol/L vs. 16.60 ± 3.05 pmol/L) levels and markedly increased TSH (10.55 ± 4.71 μIU/mL vs. 2.67 ± 1.12 μIU/mL; all $p < 0.01$), suggesting a hypothyroid trend with advancing liver disease. Comparable findings were reported by Gameel et al. (2023), who observed elevated levels of creatinine (0.7-1.35) than controls (0.6-0.9), reduced albumin levels in cases (2.56±0.48) in comparison to controls (3.95±0.67), raised AST, ALT and bilirubin levels (15-48.5, 32.5-91.5, 2.09-6.6 respectively). Similarly, the study showed lower fT3, fT4 and higher TSH levels in cirrhotic patients (0.715-3.75, 0.51-2.05, 2.68-8.35 respectively). [9]
A negative correlation is seen in cirrhotic patients between the fT3 and fT4 with the parameters of total white blood cells, bilirubin, AST, ALT, urea, creatinine and severity of cirrhosis (CTP & MELD). There is a direct relation of all above parameters with TSH. Similar relation was found by Mansour-Ghanaei et al. [10] and Yadav et al. [11].

In the present study thyroid dysfunction, particularly clinical hypothyroidism, is more prevalent as the grades of hepatic impairment increases, emphasizing its close association with disease severity. We found that out of 70 cases 55 (78.6%) demonstrated clinical hypothyroidism, whereas 15 (21.4%) showed subclinical hypothyroidism. When analyzed according to the Child–Turcotte–Pugh (CTP) classification, the majority of patients in CTP class A (n = 26) exhibited clinical hypothyroidism (73.3%), with only a single case (1.8%) of subclinical hypothyroidism. Similarly, in CTP class C (n = 43), clinical hypothyroidism remained predominant (70.9%), while subclinical hypothyroidism was noted in 7.1% of patients. These results are in agreement with the findings of Punekar P et al. lowest levels of fT3 and fT4 found were in the Child C group (1.80 ± 0.53, 1.17 ± 0.51). The study by El-Feki and Abdalla showed that patients with cirrhosis (Child–Turcotte–Pugh [CTP] class C) had a mean free T3 (FT3) level of 1.4 ± 1.0 pg/mL, whereas class B patients had fT3 of 1.9 ± 1.0 pg/mL, and class A patients had fT3 of 2.5 ± 0.6 pg/mL [12]. Thus, the thyroid profile deranges with liver impairment.

In contrast to ours, the study by Sangole SP et al. described that fT4 and TSH levels did not show significant correlations with CTP classes. They although found strong negative correlation of fT3 with CTP scores. [13]

In the present study, the mean MELD 3.0, MELD, and MELD-Na scores were 26.66 ± 6.22, 24.59 ± 6.34, and 25.26 ± 6.35, respectively. Patients with clinical hypothyroidism had higher MELD 3.0 scores (27.31 ± 6.26) compared to those with subclinical hypothyroidism (24.27 ± 5.62), indicating that worsening liver function, reflected by an elevated MELD score, was associated with more severe thyroid dysfunction. The difference was statistically significant ($p < 0.05$), suggesting a close interrelationship between hepatic insufficiency and thyroid metabolism.
These findings align with Feng et al. (2022), who who reported MELD scores of 29.57 ± 7.99 in advanced liver failure and an inverse correlation between MELD score and fT3 levels ($r = –0.430$, $p < 0.001$) in patients with liver failure, indicating that as hepatic dysfunction worsens, circulating fT3 levels decline [14]. Similarly, Ziamanesh et al. (2023) reported that fT3 can serve as a prognostic indicator of liver disease severity, with lower fT3 levels corresponding to higher MELD scores [15]. The authors attributed this to enhanced peripheral conversion of fT4 to reverse T3 via type III deiodinase activation.

In agreement, Verma et al. (2017) also found a significant inverse relationship between T3 and MELD scores, while TSH levels showed a positive correlation with MELD, reflecting compensatory hypothyroid states in progressive hepatic failure. The mean MELD score of 22.19 ± 6.64 [16]. Belu et al. (2024) further confirmed that MELD scores correlated positively with TSH at both admission and discharge and inversely with T3 levels at discharge, supporting the prognostic value of thyroid dysfunction in cirrhotic patients [17].

Moreover, the findings are consistent with evidence from Rink et al. (1991) [18] and Punekar et al. (2018), which recognized low T3 levels as sensitive predictors of disease severity and mortality in cirrhosis. Collectively, these studies and the present data emphasize that thyroid dysfunction, especially reduced T3 levels and increased TSH, parallels the progression of liver failure and can complement MELD scoring in prognostication.

Conclusion

This study showed that thyroid hormone levels, fT3 and fT4 results in decline while there is significant increase in TSH in the patients suffering from liver cirrhosis in comparison to those with no comorbidities. Also, the dysfunction of these hormone levels is positively associated with the severity of cirrhosis, computed using the CTP, MELD, MELD 3.0 and MELD sodium scores.

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