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Original Research Article

Thyroid hormone dysfunction in liver cirrhosis

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ABSTRACT

Background: Chronic liver disease (CLD) relentlessly progresses to fibrosis and cirrhosis unless the causative agent is eliminated. Cirrhosis leads to hepatic dysfunction and additionally has an effect on several systems. Liver is vital to thyroid hormone metabolism and also in the synthesis of thyroid binding globulin. Low free T3 syndrome is described in liver cirrhosis. Aim was to investigate the thyroid function of patients with CLD in relation to the severity of the disease.
Methods: This is a prospective observational, single centre, hospital-based case-control study, comparing thyroid function in 112 decompensated CLD subjects and 112 matched healthy controls.
Results: In the present study majority of participants are between the ages of 31 to 50 years. TSH level was found to be elevated in hepatic encephalopathy (HE) patients with a higher West Haven grade compared to lower grades of encephalopathy. Reduced levels of FT3 and FT4 were found in higher grades of HE. MELD score and Child Pugh score severity correlation with FT3 has shown a reduced FT3 and was directly proportional to the severity of CLD.
Conclusions: Patients with CLD showed abnormalities in thyroid hormone concentrations and these abnormalities were related to advanced Child Pugh score and MELD score. Serum T3 level has been found to be low in significant number of cirrhotic patients.

Keywords: Thyroid function, TSH, Free T3, Chronic liver disease, West Haven score, MELD score, Child Pugh score

INTRODUCTION

Chronic liver disease (CLD) is a progressive condition that involves ongoing inflammation, damage to liver cells, and attempts at regeneration, which can eventually lead to fibrosis and cirrhosis.[1] The liver is crucial for metabolizing, transporting, and activating thyroid hormones, and it also plays a key role in producing thyroid-binding globulin. The liver and thyroid have a significant impact on each other's functions, whether in a healthy state or during illness. Cirrhosis of liver is categorized as either "compensated" or "decompensated".[2] Decompensation includes Jaundice, Ascites, HE or Variceal bleed. Other signs, such as hepatorenal syndrome, low sodium levels (hyponatremia), and spontaneous bacterial peritonitis, can also indicate decompensation, although ascites usually appears first.

The two major hormones produced by thyroid gland are thyroxine (T4) and triiodothyronine (T3).[3] In individuals with CLD, levels of these thyroid hormones and their binding proteins often change.[4] A common endocrine condition seen in cirrhosis is the "low T3 syndrome," which features lower serum T3 levels, higher reverse T3 (rT3), and a decreased T3:T4 ratio. This condition is believed to be an adaptive response that helps lower the basal metabolic rate of liver cells and preserve liver function.

HE is one of the serious decompensations of liver disease, leading to changes in mental status, cognitive difficulties,

and motor issues.[5] Hypothyroidism can cause similar neuropsychiatric symptoms, so it's important to consider it when diagnosing encephalopathy in patients with CLD. The Child-Pugh score, (also known as the Child-Turcotte-Pugh (CTP) classification), is a widely used tool for evaluating the prognosis of patients with cirrhosis. It divides patients into three categories: Class A (well-compensated), class B (moderate dysfunction), and class C (severe hepatic impairment).

The current study focuses on the thyroid profile in CLD in relation to the severity of the disease, mortality and also to study the relationship between serum FT3 and Child-Pugh score.

METHODS

Research design

It is a prospective observational study.

Study sample

In this study 112 decompensated CLD patients were compared with 112 healthy controls.

Sample collection and analysis of thyroid hormones

A directly labeled antibody, competitive technique is used. FT3/FT4 present in the sample competes with ligand on the modified well surface for a limited number of binding sites on a horseradish peroxidase (HRP)-labeled antibody conjugate (sheep anti-T3). The well surface was modified and coated to act as a ligand for unconjugated component. Excess unbound material was eliminated by washing. The assay was designed with optimal reagent concentrations to ensure that disruption of the T3/binding protein equilibrium or T4/binding protein equilibrium is minimal and insignificant. Detection of bound HRP conjugate was done by a luminescent reaction. A reagent mature included luminogenic substrates (a luminol derivative and a peracid salt) and an electron transfer agent was added to each well. The HRP in the bound conjugate catalyzes the oxidation of the luminol derivative, results in light emission. The electron transfer agent (a substituted acetanilide) amplifies light signal produced and extends its emission. The light output is measured by the system. The signal intensity is indirectly proportional to concentration of FT3/FT4 in the sample.

TSH measurement was carried out by using the VITROS TSH Reagent Pack and the Vitros TSH Calibrators on the VITROS 5600 Integrated System using Intellicheck® Technology. It is an immunoassay technique resulting in formation of antigen -antibody complex where TSH present in the sample reacts with a biotinylated antibody (mouse monoclonal anti-whole TSH) and a horseradish peroxidase (HRP)-labeled antibody conjugate (mouse monoclonal anti-TSH β-subunit). This complex is captured by streptavidin on the wells. Unbound substances are cleared by washing while the bound HRP conjugate was estimated by a chemiluminescent reaction. The HRP in the bound conjugate catalyzes the oxidation of the luminol derivative, producing light. The electron transfer agent (a substituted acetanilide) amplifies the level of light produced and extends the emission. The light signals are read by the systems. The amount of HRP conjugate bound is directly proportional to the concentration of TSH present.

Inclusion criteria

The study included male and female subjects with known diagnosis of cirrhosis on the basis of clinical, radiological (abdominal ultrasound) and biochemical studies, between 18 to 80 years of age. All patients underwent a clinical examination according to the predetermined proforma and signed appropriate informed consent.

Control sample

Age and sex matched hospital health care employees with no history of liver disease or other comorbidities were considered as control group after obtaining informed and written consent.

Exclusion criteria

Patients with known cases of hypothyroidism and undergoing treatment, pregnant women, patients with sepsis, and patients using medications that interfere with thyroid metabolism, such as amiodarone, phenytoin, NSAIDS, and salicylates were excluded.

Study site

It is a single-centre, hospital-based study conducted from September 2021 to August 2022 in the Department of Gastroenterology, Yashoda Hospital, Secunderabad, and approved by the institutional ethical committee.

After a thorough history of the patient's illness, including liver cirrhosis and thyroid disease, general examination was conducted. Complete blood count (CBC), serum creatinine, serum albumin, serum sodium, bilirubin, PT/INR, T3, T4, TSH were estimated. Decompensated CLD (DCLD) severity grading was determined using two scales, namely the Child Pugh score and the MELD score. Thyroid abnormalities like low FT3, low FT4, and high TSH were compared on these scales. Electrochemiluminescence immunoassay was used to perform thyroid function tests (TFT). The normal range of TSH is between 0.35 and 5.5 µIU/ml, FT4 is between 0.8 and 2.7 ng/dl, and FT3 is between 2.1 and 4.4 pg/ml.

RESULTS

Table 1 shows distribution of age groups and gender of the individuals of the study. A total of 224 individuals were examined of which 112 were cirrhotic patients and the

remaining were healthy controls. The mean age distribution for cases and controls was found to be 43±13 years and 40±14 years, respectively (p=0.1). The subjects were majorly between the ages of 31 and 40 for controls (39.3%) and 41 to 50 for cases (33.9%). Of the 112 cases in test group, 108 were men and 4 were women (p=0.33).

The thyroid profile of the total studied population is presented in Table 2, with low free T3 (37.5%, p<0.001), low free T4 (10.7%, p=0.17), and high TSH (16.1%, p=0.74) being the most frequently observed abnormalities. Prevalence of hypothyroidism (TSH elevation) was observed in 10.7% of controls (12 out of 112 controls) compared to 16.1% in the test group (18 out of 112 cases).

Table 3 depicts the variation in the thyroid profile of the cases based on West Haven score for HE. The TSH level was higher by 29% from Grade 0 to grade 3 whereas FT3 and FT4 were lower by 4% and 1% respectively. The FT3 values have shown an average decrease of 8% from the normal range.

Table 4 explains the survival rate of the cirrhosis cases with reference to West Haven score. Results also show that, with the increase in Haven score, the non survivors (deaths) with abnormal TSH, FT3, FT4 levels increased. In case of the survivors the increase in the Haven grade explains that, there is not much variation in the thyroid profile but slight variation is reported in FT3. 33% of cirrhosis non-survivors (Deaths) with HE has TSH abnormality which is statistically insignificant (p=0.07). Low FT3 levels were seen in 75% of cirrhosis cases (non survivors) with HE whereas in HE survivors it is 33.3 % which is statistically significant (p=0.004).

Table 5 shows the comparison of thyroid profile among cases based on Pugh score. In comparison, FT4 levels were found to be least affected followed by FT3 and TSH. When the mean serum level of FT3 was compared, Child C group (1.80±0.53) had the lowest levels followed by Child B group (2.20±0.55) whereas Child A group (2.19±0.5) had the highest levels.

Results also show that as the MELD score increases, the severity of low FT3 increases significantly at p=0.03 and raised TSH at p=0.72, low FT4 at p=0.06 also increases (Table 6). Present results also show that the FT3 and FT4 levels were reduced by 65.2% and 40 % with the increase in MELD score.

TSH levels were in positive correlation with bilirubin, globulin, PT, serum creatinine, and severity of liver disease (CTP and MELD score) in all cirrhotic patients, while FT3 and FT4 were negatively correlated (Table 7).

Table 1: Total study population distribution based on gender and age.

Variables	Case	Control
Gender		
Female	04	12
Male	108	100
Age (in years)		
23-30	0	04
31-40	24	44
41-50	38	24
51-60	32	24
>60	18	12

Table 2: Total Study population distribution based on thyroid hormones.

Study population	TSH, (n=224)		FT3, (n=224)		FT4, (n=224)	
	Abnormal	Normal	Abnormal	Normal	Abnormal	Normal
Case	18	94	42	70	12	100
Control	12	100	0	112	0	112

Table 3: Thyroid profile of cases based on West Haven classification (HE).

West Haven score	Total no. cases for every studied parameter	TSH, (n=112) (p=0.11) (µIU/ml)	FT3 (n=112), (p=0.009) (pg/ml)	FT4 (n=112), (p=0.17) (ng/dl)
0	76	3.58±1.52	1.72±0.72	1.13±0.68
1	02	4.56±1.25	1.68±0.68	1.25±0.54
2	14	5.08±1.62	1.66±0.85	1.15±0.65
3	20	4.65±1.35	1.65±0.75	1.12±0.86
4	0	-	-	-

Table 4: Distributions of cases based on HE.

Patient condition	West Haven score	TSH, (p=0.07)		FT3, (p=0.004)		FT4, (p=0.18)	
		Abnormal	Normal	Abnormal	Normal	Abnormal	Normal
Death	2	0	6	02	04	0	06
	3	08	10	16	02	06	12
Survivors	1	0	02	02	0	0	02
	2	0	08	02	06	0	08
	3	0	02	0	02	0	02

Table 5: Thyroid profile of cases based on Child Pugh score.

Child Pugh score	No. patients studied for each parameter, (n=112)	TSH, (mean±SD) (p=0.67) (µIU/ml)	FT3, (mean±SD) (p=0.06) (pg/ml)	FT4, (mean±SD (p=0.71) (ng/dl)
A	06	2.43±1.34	2.19±0.5	1.76±0.71
B	42	3.09±1.34	2.20±0.55	1.75±0.72
C	64	3.41±1.62	1.80±0.53	1.72±0.61

*TSH: Thyroid stimulating hormone, FT3: Free triiodothyronine, FT4: Free thyroxine, SD: Standard deviation

Table 6: Thyroid profile of cases based on MELD score.

Thyroid profile	MELD score	
	≤20 (mean±SD)	>20 (mean±SD)
TSH, (n=112) (p=0.72) (µIU/ml)	1.5±0.2	5.8±0.3
FT3, (n=112) (p=0.03) (pg/ml)	4.3±0.1	1.5±0.1
FT4, (n=112) (p=0.66) (ng/dl)	3.0±0.3	1.8±0.2

*TSH: Thyroid stimulating hormone, FT3: Free triiodothyronine, FT4: Free thyroxine, SD: Standard deviation, MELD: Model for end stage liver disease.

Table 7: Relationship between various parameters and thyroid hormone levels in cases with liver cirrhosis.

Variables	Mean±SD	TSH		FT3		FT4	
		R	P	R	P	R	P
Serum creatinine	1.76±1.05	0.44	0.04	-0.10	0.008	-0.24	0.04
Bilirubin	8.63±11.80	0.23	0.3	-0.33	<0.0001	-0.29	<0.0002
PT INR	1.90±0.32	0.06	0.003	-0.33	0.06	-0.03	0.64
Serum sodium	129.11±3.06	0.19	0.94	0.26	<0.0001	0.17	<0.0001
Serum albumin	2.51±0.64	0.08	0.31	-0.07	<0.001	-0.29	<0.0004
MELD score	24.22±8.21	0.31	0.06	-0.63	0.03	-0.45	0.66
CTP score	4.18±0.58	-0.176	0.74	-0.57	<0.001	-0.191	0.17

*TSH: Thyroid stimulating hormone, FT3: Free triiodothyronine, FT4: Free thyroxine, SD: Standard deviation, r=correlation coefficient, p=significance level.

DISCUSSION

Most cases of cirrhosis and chronic liver failure that could be prevented are linked to excessive alcohol use, viral hepatitis, or non-alcoholic fatty liver disease (NAFLD). These causes are among the top global contributors to illness and death. Often, cirrhosis develops slowly, with many patients not showing any symptoms until they reach a critical stage. The common manifestations of decompensated cirrhosis include variceal bleeding, spontaneous bacterial peritonitis, ascites, and/or HE.[6]

In this study conducted at a single center, we noted a clear predominance of male patients suffering from decompensated CLD. The most common symptom reported was abdominal distension, followed by fatigue, confusion, disorientation, hematemesis, and/or melena. The age distribution of the patients in this study aligned with findings from earlier research.[7]

To assess the severity of decompensated CLD, the Child-Pugh and MELD (Model for End-stage liver disease) scores were employed. Thyroid function tests, including TSH, FT4, and FT3 levels, were analyzed alongside these scores to evaluate thyroid dysfunction. The study found that abnormal thyroid profiles were linked to the severity of liver disease. Specifically, a rise in MELD score was significantly associated with lower FT3 levels, while elevated TSH and decreased FT4 levels did not show a statistically significant relationship.[8] However, Child-Pugh and MELD scores is negatively correlated with T3 levels.

Various studies revealed that there is a confirmed association between serum creatinine and TSH levels.[9] In patients with over hypothyroidism, FT3 and FT4 levels were negatively correlated with liver enzymes, while TSH levels had a positive association.[10] Patients with decompensated non-alcoholic cirrhosis, having HE are typified by low FT3 and low total T4. All cirrhotics had significantly low FT3 levels.[9] Some patients with CLD may have thyroiditis, hyperthyroidism, or hypothyroidism.[6] Deviations in levels of thyroid hormone in patients with cirrhosis like low serum T3 levels, elevated rT3 (inactive form), and normal level of TSH are often observed. There could be multiple reasons behind these irregularities which include reduced conversion of T4 to T3, decreased hepatic clearance of rT3, altered binding of T4 and T3 to their carrier protein, and altered plasma level of thyroid binding proteins.[11] Cirrhosis patients have reduced conversion of T4 to T3 and is caused by inhibition of type 01 deiodinase enzymes. Type 02 deiodinase enzyme if active, the majority of T4 is transformed into rT3, which raises rT3. The current study also identified a significant link between low FT3 syndrome and TSH levels in CLD patients. All cirrhotic patients showed significantly reduced FT3 levels, reinforcing the connection between liver status and thyroid function.

CONCLUSION

In conclusion, patients with decompensated liver cirrhosis have several abnormalities in thyroid profile and serum level of T3 is low in a significant number of patients. Patients with liver cirrhosis should have regular thyroid function assessment to reduce the morbidity and mortality.

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