

Original Research Article

A STUDY OF THYROID DYSFUNCTION IN DECOMPENSATED CIRRHOSIS OF LIVER AND CORRELATION WITH ETIOLOGY OF CIRRHOSIS AND SEVERITY OF UNDERLYING LIVER DISEASE

Rohan Ghosh¹, Souvik Basu², Arpan Chakrabarti³

¹Senior Resident, Department of General Medicine, Calcutta National Medical College and Hospital, Kolkata, West Bengal, India.

²Assistant Professor, Department of General Medicine, Burdwan Medical College and Hospital, Burdwan, West Bengal, India.

³Senior Resident, Department of General Medicine, Burdwan Medical College and Hospital, Burdwan, West Bengal, India.

Received : 22/08/2026
Received in revised form : 20/09/2026
Accepted : 03/10/2026

Corresponding Author:

Dr. Souvik Basu,

Assistant Professor, Department of General Medicine, Burdwan Medical College and Hospital, Burdwan, West Bengal, India.

Email: sbasu.pg@gmail.com

DOI: 10.70034/ijmedph.2026.4.70

Source of Support: Nil,

Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (4); 480-486

ABSTRACT

Background: Liver cirrhosis is often associated with altered thyroid function, which may reflect the severity of liver dysfunction. Understanding this relationship can help in predicting disease progression and prognosis in cirrhotic patients. The aim of this study was to study of thyroid dysfunction in decompensated cirrhosis of liver and correlation with etiology of cirrhosis and severity of underlying liver disease.

Materials and Methods: The present Institution based descriptive observational cross sectional study was carried in the Department of General Medicine of Calcutta National Medical College and Hospital, Kolkata. 113 Known case of Thyroid disorder or Family history of Thyroid disorder fulfilling the inclusion criteria were included in the study. The research was carried out between January 2020 to June 2021. The statistical data were analyzed using SPSS V.25.0 and Microsoft Excel.

Results: Mean age of the patients was 56.23±11.21 years. Cirrhosis of liver was more prevalent among males (79%) compared to females (21%). During study, gross ascites (73.5%) was found most common presentation. There was a significant difference regarding the etiology of liver cirrhosis and thyroid profile. Patient with low T3 levels were highest in Child-Pugh class C (82.1%) followed by Child-Pugh class B (61.9 %) and Child-Pugh class A (50%) and this difference was found to be statistically significant (p=0.037). Patients with low FT4 level having MELD score of more than 20 were significantly higher as compared to the patients with MELD score less than 20.

Conclusions: Thyroid abnormalities are not uncommon in patients with cirrhosis. Hypothyroidism is the most common abnormality that was detected. Thyroid function test should be carried out in all cirrhotic patients to assess the severity and prognostication of such patients.

Keywords: Liver cirrhosis, Thyroid dysfunction, T3, FT3, TSH, Hypothyroidism, Liver dysfunction.

INTRODUCTION

Cirrhosis is the leading cause of liver-related death globally.^[1] It is the endstage of progressive liver fibrosis, in which the hepatic architecture is distorted.^[2] Decompensation in patients with compensated cirrhosis is usually defined as the first occurrence of ascites, esophageal variceal bleeding,

hepatic encephalopathy, and, in some individuals, increased bilirubin concentration.^[3,4]

Once decompensation occurs, the mortality and morbidity resulting from cirrhosis increase sharply, and depending on the cause of decompensation, the 1-year case-fatality rate can be as high as 80%.^[5,6]

Almost all of the mortality and morbidity resulting from cirrhosis is caused by the decompensated type. Such patients need frequent medical attention and an

increasing amount of medication over the disease course. Quality of life is affected and frequent hospitalisations (admissions and stays) are required.^[7]

As the disease progresses, hospital stays become more frequent and more prolonged. Finally, patients either die or receive a liver transplant, which is a high-burden option for patients, health-care systems, and health financing and governance.^[7]

The diagnosis of cirrhosis depends on demonstrating widespread nodules in the liver combined with fibrosis. So, liver biopsy is the gold standard for diagnosis of cirrhosis.^[8]

Hepatorenal syndrome, hyponatremia, and spontaneous bacterial peritonitis are also features of decompensation, but in these patients, ascites invariably occurs first. None of these characteristics are present in compensated cirrhotic individuals.^[9]

Thyroxine (T4) and triiodothyronine (T3) are two hormones produced by the thyroid gland. These hormones, which act through thyroid hormone receptors are important for cell differentiation throughout development and for maintaining thermogenic and metabolic homeostasis in adults.^[10]

The liver is the most essential organ in the peripheral conversion of tetra-iodothyronine (T4) to tri-iodothyronine (T3) by Type 1 deiodinase, which is crucial for thyroid hormone metabolism.^[11,12] Type I deiodinase is the most important enzyme in the liver, accounting for 30–40% of extra-thyroidal T3 synthesis. It may deiodinate T4 to T3 in both 5' and 5 directions. The liver is also involved in thyroid hormone conjugation and excretion, as well as thyroid binding globulin production.^[13] T4 and T3 affect hepatic function through regulating the baseline metabolic rate of all cells, including hepatocytes. The liver metabolizes thyroid stimulating hormone (TSH) and controls their endocrine effects throughout the body. Thyroid disorders can impact liver function, while liver disease can affect thyroid hormone metabolism. A number of systemic diseases can affect both organs.^[14]

TSH levels in the blood, on the other hand, remain normal or slightly elevated. Thyroid hormone levels have been shown to vary in such a way that some researchers have proposed for it to be used as a sensitive indicator of liver function.^[15,16]

The present study was conducted with the objective to identification of different types of thyroid

abnormalities in patients with decompensated cirrhosis of liver and to correlate thyroid abnormalities with etiology and severity of cirrhosis.

MATERIALS AND METHODS

The present Institution based descriptive observational cross sectional study was carried in the Department of General Medicine in collaboration with Department of Biochemistry, Pathology, Sonography and Endoscopy Unit of Department of General Medicine of Calcutta National Medical College and Hospital, Kolkata. The study was carried out from January 2020 to June 2021.

Inclusion Criteria: All patient >12yrs with Symptoms, Signs, Biochemical and Radiological features of Decompensated Cirrhosis of Liver irrespective of cause. Patients who were willing to part of study after consent

Exclusion Criteria: Known case of Thyroid disorder or Family history of Thyroid disorder with Cirrhosis of Liver and Patients with Sepsis, Cardiac failure, Pancreatitis, CKD, Nephrotic syndrome, Pregnancy.

Sample size: 113 patients fulfilling the inclusion criteria were included in the study. A predesigned, pretested, semi-structured, interviewer-administered questionnaire was used.

Study Technique: Data was collected by History, Clinical examination, Appropriate investigation and recorded in a preformed proforma. For each patient thorough history, clinical examination and investigations was done. Child-Turcotte-Pugh (CTP) Score and Model for End-Stage Liver Disease (MELD) Score was calculated. Results were analysed for etiology and severity of liver disease and compared with Fasting Thyroid Profile.

Data analysis: All Statistical Analysis Plan was done using IBM SPSS (Statistical Package for the Social Sciences) Version 21. Data was presented as mean $\pm$ SD or number (%) unless specified. All parametric data was analysed using Student's t-test. All non-parametric data was analysed by Chi-square test. A p-value of <0.05 was considered statistically significant.

Ethical issues

Institutional Ethics Committee of the Calcutta National Medical College & Hospital, Kolkata approved the study.

RESULTS

Table 1: Distribution according to socio-demographic profile and incidence of etiology. (n=113)

Socio-demographic profile		Number	Frequency (%)
Age Group (years)	<18 years	0	0.0
	18-35 years	17	15.0
	36-50 years	63	55.8
	51-65 years	20	17.7
	>65 years	13	11.5
Total		113	100.0

	Mean Age	56.23±11.21	
Gender	Male	89	78.8
	Female	24	21.2
Etiology	Alcoholism	72	63.7
	HBV	22	19.5
	NASH	13	11.5
	HCV	6	5.3

Majority of patients 63 (55.8%) belonged to age group 36-50 years, 20 (17.7%) patients belonged to age group 51-65 years, 17 (15%) patients belonged to the age group 18-35 years and the least common age group was above 65 years with 13 (11.5%) patients with a mean age of 56.23±11.21 years. Cirrhosis of liver was more prevalent among males (79%)

compared to females (21%). Most common etiology of liver cirrhosis was Alcoholism which comprised 72 (63.7%) patients, followed by HBV related cirrhosis comprised 22 (19.5%) patients, NASH comprised 13 (11.5%) patients and 6 (5.3%) patients had HCV related cirrhosis. (Table 1)

Table 2: Distribution according to clinical manifestations

Clinical Manifestations	Frequency	Percentage
Ascites	83	73.5
Anemia	75	66.4
Coagulation Abnormality	73	64.6
Thrombocytopenia	60	53.1
Constipation	55	48.7
Hepatic Encephalopathy	43	38.1
Upper gastrointestinal bleed	38	33.6
Jaundice	36	31.9
Sepsis	25	22.1
Azotemia	19	16.8
Pleural Effusion	18	15.9
Shock	15	13.3

During study, gross ascites (73.5%) was found most common presentation and other complications were anemia (66.4%), thrombocytopenia (53.1%), coagulation abnormality (64.6%), constipation (48.7%), HE (38.1%), jaundice (31.9%), upper gastrointestinal bleed (33.6%), azotemia (16.8%), sepsis (22.1%), pleural effusion (15.9%), and shock (13.3%). (Table 2)

Table 3: Distribution according to Child Pugh Score, MELD score and Prevalence of Thyroid Dysfunction

Child Pugh Score	Frequency	Percentage
A	4	3.5
B	42	37.2
C	67	59.3
MELD score		
≤ 20	71	62.8
>20	42	37.2
Thyroid Dysfunction		
Euthyroid	77	68.2
Hyperthyroidism	4	3.5
Hypothyroidism	32	28.3

Based on Child Pugh score, 4 patients (3.5%) were classified as Child-Pugh class A, 42 patients (37.2%) as Child-Pugh class B and rest of the 67 patients (59.3%) as Child-Pugh class C. Based on MELD score, 71 (62.8%) and 42 (37.2%) patients were

found to be less than and more than 20 MELD score respectively. Out of 113 patients 32 (28.3%) patients had hypothyroidism, 4 (3.5%) patients had hyperthyroidism and 77 (68.2%) patients had normal thyroid profile. (Table 3)

Table 4: Association between Thyroid dysfunction and Etiology

Etiology	Euthyroid(n=77)		Hyperthyroidism(n=4)		Hypothyroidism(n=32)		Total
	Frequency	%	Frequency	%	Frequency	%	
Alcoholism	43	55.84	1	25	28	87.50	72 (63.72)
NASH	20	25.97	1	25	1	3.13	22 (19.47)
HBV	10	12.99	1	25	2	6.25	13 (11.50)
HCV	4	5.19	1	25	1	3.13	6 (5.31)
Total	77	100.0	4	100.0	32	100.0	113 (100.0)
Statistical Inference	Chi-square-29.0536 P Value-0.0001**						

Table 4 presents the data regarding the association between thyroid function and etiology of liver cirrhosis. From above analysis we found there was a

significant difference regarding the etiology of liver cirrhosis and thyroid profile (p value = <0.0001).

Table 5: Correlation between Total T3 level, FT4 level, TSH level and Child Pugh Score

TotalT3 level	ChildPugh ClassA(n=4)		ChildPughClassB (n=42)		ChildPughClass C(n=67)		Total	Statistical Inference
	No.	%	No.	%	No.	%		
Low	2	50.0	26	61.9	55	82.1	83 (73.5)	Chi-square-6.5634 P Value-0.037*
Normal	2	50.0	16	38.1	12	17.9	30 (26.5)	
High	0	0.0	0	0.0	0	0.0	0 (0.0)	
Total	4	100.0	42	100.0	67	100.0	113 (100.0)	
FT4level								Chi-square-14.9484 P Value-0.004*
Low	1	25.0	21	50.0	40	59.7	62 (54.9)	
Normal	2	50.0	20	47.6	27	40.3	49 (43.4)	
High	1	25.0	1	2.4	0	0.0	2 (1.8)	
Total	4	100.0	42	100.0	67	100.0	113(100.0)	
TSHlevel								Chi-square-11.9615 P Value-0.017*
High	0	0.0	9	21.4	23	34.3	32 (28.3)	
Normal	3	75.0	30	71.4	44	65.7	77 (68.1)	
Low	1	25.0	3	7.1	0	0.0	4 (3.5)	
Total	4	100.0	42	100.0	67	100.0	113(100.0)	

It shows that patient with low T3 levels were highest in Child-Pugh class C (82.1%) followed by Child-Pugh class B (61.9 %) and Child-Pugh class A (50%) and this difference was found to be statistically significant (p=0.037). Patient with low free T4 levels were highest in Child-Pugh class C (59.7%) followed by Child-Pugh class B (50 %) and Child-Pugh class

A (25%) and this difference was found to be statistically significant (p=0.004). Patient with high TSH levels were highest in Child-Pugh class C (34.3%) followed by Child-Pugh class B (21.4 %) and none of the patients in Child-Pugh class A has high TSH level and this difference was found to be statistically significant (p=0.017). (Table 5)

Table 6: Correlation between Total T3 level, FT4 level, TSH level and MELD Score

Total T3 level	MELDScore≤20 (n=71)		MELD Score >20(n=42)		Total	Statistical Inference
	No.	%	No.	%		
Low	46	64.8	37	88.1	83 (73.5)	Chi-square-7.3509 P Value-0.006*
Normal	25	35.2	5	11.9	30 (26.5)	
High	0	0.0	0	0.0	0 (0.0)	
Total	71	100.0	42	100.0	113 (100.0)	
FT4level						Chi-square- 7.8972 P Value-0.019*
Low	32	45.1	30	71.4	62 (54.9)	
Normal	37	52.1	12	28.6	49 (43.4)	
High	2	2.8	0	0.0	2 (1.8)	
Total	71	100.0	42	100.0	113 (100.0)	
TSHlevel						Chi-square- 3.8031 P Value-0.149(NS)
Low	17	23.9	15	35.7	32 (28.3)	
Normal	50	70.4	27	64.3	77 (68.1)	
High	4	5.6	0	0.0	4 (3.5)	
Total	71	100.0	42	100.0	113 (100.0)	

It shows that patients with low T3 level having MELD score of more than 20 were significantly higher as compared to the patients with MELD score less than 20. Patients with low FT4 level having MELD score of more than 20 were significantly higher as compared to the patients with MELD score less than 20. Statistically no significant difference was found between TSH levels and MELD score (p value=0.149). (Table 6)

DISCUSSION

Cirrhosis is defined as the histological development of regenerative nodules surrounded by fibrous bands in response to chronic liver injury that leads to portal hypertension and end stage liver disease. In clinical

terms, cirrhosis is described as either compensated or decompensated. Decompensation means cirrhosis complicated by one or more of the following features: jaundice, ascites, hepatic encephalopathy (HE), or bleeding varices. Ascites is the usual first sign.^[9] In industrialized countries chronic hepatitis C and alcoholism are still two leading causes of cirrhosis.^[17] Autoimmune hepatitis, metabolic liver disease, Wilson's disease and primary biliary cirrhosis are among other causes of liver cirrhosis. Hence keeping an eye in the present scenario, the present study was conducted with the aim to identify different types of thyroid abnormalities in patients with decompensated cirrhosis of liver and to correlate thyroid abnormalities with etiology and severity of cirrhosis.

Majority of patients in the present study i.e. 63 (55.8%) patients were belonged to age group 36-50 years, 20 (17.7%) patients were belonged to age group 51-65 years, 17 (15%) patients were belonged to the age group 18-35 years and the least common age group was above 65 years with 13 (11.5%) patients with a mean age of 56.23 ± 11.21 years. The observation of the present study also reveals that cirrhosis of liver was more prevalent among males (79%) compared to females (21%).

Mansour-Ghanaei F et al (2012) in their study reported a total of 64 patients (42 male and 22 female) with a mean age of 55.03 ± 12.05 were enrolled for their study.^[18]

Sudhir Kumar Verma et al (2017) in their study reported based on inclusion criteria, 102 patients were enrolled in their study in which 73 were males and 29 were females with a mean age of 41 ± 13.7 years.^[19]

During study, gross ascites (73.5%) was found most common presentation and other complications were anemia (66.4%), thrombocytopenia (53.1%), coagulation abnormality (64.6%), constipation (48.7%), HE (38.1%), jaundice (31.9%), upper gastrointestinal bleed (33.6%), azotemia (16.8%), sepsis (22.1%), pleural effusion (15.9%), and shock (13.3%).

Punekar et al (2018) also reported the same. In their study ascites (74%) was found most common presentation and other complications were anemia (87%), thrombocytopenia (53%), coagulation abnormality (65%), HE (38%), jaundice (32%), upper gastrointestinal bleed (34%), azotemia (17%), pleural effusion (16%), sepsis (22%), shock (14%). Constipation was found in 49% patients.^[20]

In our studies, most common etiology of liver cirrhosis was Alcoholism which comprised 72 (63.7%) patients, followed by HBV related cirrhosis comprised 22 (19.5%) patients, NASH comprised 13 (11.5%) patients and 6 (5.3%) patients had HCV related cirrhosis.

Samarthana V et al (2020) in their reported most common etiology of liver cirrhosis was Alcoholism which comprised 85 (85%) patients, 8 (8%) patients had HBV related cirrhosis, and 7 (7%) patients had HCV related cirrhosis.^[21]

Punekar et al (2018) in their study reported alcohol was most common etiology (46%) followed by hepatitis B (19%), hepatitis C (3%), Wilson disease (1%), and others.^[20]

Regarding the severity of the cirrhosis of liver we found 4 patients (3.5%) were classified as Child-Pugh class A, 42 patients (37.2%) as Child-Pugh class B and rest of the 67 patients (59.3%) as Child-Pugh class C. Based on MELD score, 71 (62.8%) and 42 (37.2%) patients were found to be less than and more than 20 MELD score respectively.

Similar observation was reported by Sudhir Kumar Verma et al (2017). In their study based on CP score, 4 patients (3.92%) were classified as Child-Pugh class A, 40 patients (39.22%) as Child-Pugh class B and rest of the 58 patients (56.86%) as Child-Pugh

class C and according to MELD score 64 (62.75%) and 38 (37.25%) patients were found to be less than and more than 20 MELD score respectively.^[19]

Asish Kumar et al (2020) in their study reported severity of cirrhosis using Child-Turcotte-Pugh (CTP) criteria. On assessment of severity of cirrhosis (52%) were belonged to CTP A, (38%) to CTP B and (10%) to CTP C.^[22]

While analyzing the thyroid profile of the study subjects we found, out of 113 patients 32 (28.3%) patients had hypothyroidism, 4 (3.5%) patients had hyperthyroidism and 77 (68.2%) patients had normal thyroid profile. Out of 77 euthyroid participants alcoholism was the most common etiology comprised of (55.84%) participants followed by non-alcoholic steatohepatitis (25.97%). Among patients with hypothyroidism also alcohol was the most common etiology (87.5%) followed by HBV (6.25%). From above analysis we found there was a significant difference regarding the etiology of liver cirrhosis and thyroid profile (p value = <0.0001).

Asish Kumar et al (2020) in their study reported biochemical hypothyroidism was seen in 5 out of 50 patients (10%) while hyperthyroidism was observed in 2 cases (4%). Among the patients with biochemical hypothyroidism (elevated TSH) only one patient had low T3 while all had normal T3 and T4 levels. Among the patients with hypothyroidism, 3 (60%) had ethanol related liver cirrhosis, 1 (20%) had Hepatitis C whereas 1 had cryptogenic cirrhosis (20%). On the other hand 3 out of 21 patients (14.28%) with ethanol related liver cirrhosis, 1 out of 20 patients (5%) with Hepatitis C related liver cirrhosis and 1 out of 4 patients (25%) with cryptogenic cirrhosis had hypothyroidism.^[22]

Kalmani VM et al (2019) in their study reported that hypothyroidism (increase in TSH) was seen in 34 % of the patients i.e., 17 out of 50 patients.^[23] This was in comparison with 16 % of the patients in Kharb S et al,^[23] (2015) . 20 % of the patients in P Punekar et al,^[20] (2018) and 21.6 % of the patients in Joieimon et al,^[24] (2017) which was almost similar to our study. When compared to the study done by Samarthana V et al. (2020) they noticed normal TSH levels in 36 % of the patients studied and an increase in the TSH levels in 62 % among the cirrhotics of the study group.^[21]

The present study was conducted with the aim to identify different types of thyroid abnormalities in patients with decompensated cirrhosis of liver. While analysing the above we found the most common thyroid abnormality was low T3 level found in 83 (73.5%) patients. The low T3 syndrome has frequently been reported in patients with chronic liver disease.^[25] We also observed low T3, FT4 and slightly raised TSH in most of our enrolled patients.

In several studies, the most common abnormalities of serum thyroid hormone concentration in cirrhotic patients observed were, low serum T3 level, raised T3 level, and a normal TSH levels. There are

many factors those may be responsible for these abnormalities, which includes alteration in plasma level of thyroid binding proteins, altered binding of T4 and T3 to their carrier protein, impaired hepatic clearance of reverse T3 (rT3), hyperglucagonemia, and reduced extrathyroidal conversion of T4 to T3. In cirrhotic patients, because of extensive hepatic inflammation and fibrosis, there is inhibition of Type 1 deiodinase (D1) enzymes that lead to decreased conversion of T4 to T3. Since the type 2 deiodinase (D2) enzymes remain active, now most of the T4 is converted into rT3 leading to increased rT3 levels.^[26] In a small study Borzio et al(1983) reported significantly low T3, fT4, T3/thyroxine binding globulin, T4/thyroxine binding globulin ratios and TSH, while elevated rT3 and thyroxine binding globulin.^[27] Joeimon et al(2017) from India demonstrated high incidence of subclinical hypothyroidism (21.6%) characterized by elevated TSH and low T3 in 50% of the patients with hypothyroidism. Remaining 50% of hypothyroid patients had normal T3, fT4 and raised TSH.^[25]

Kalmani VM et al (2019) in their study reported the common abnormality among the 50 patients studied noted that there was a decrease in T3 in 52% of the patients i.e., 26 out of 50 patients.^[24] It was in comparison with a study done by Mobin et al.(2016) where they noted a decrease in T3 levels in 76.3 % of the patients.^[28] This was also seen in the study done by Kharb S et al,^[26] (2015).

In the present study we observed an inverse correlation between T3 level and CTP score and MELD score. The severity of the liver disease correlated with the low levels of the T3 hormone in the form of CTP or MELD. With a rise in the MELD score, the level of TSH also increases. This change in the levels of thyroid hormones is one of the prognostic markers in patients suffering from cirrhosis. The liver is responsible for the peripheral conversion of tetra-iodothyronine (T4) to tri-iodothyronine (T3) by type-1 deiodinase.^[11,26] There is 30 - 40 % of type-1 deiodinase in the liver accounting for extra thyroidal production of T3.^[11] The thyroid hormones are >99% bound to thyroxine binding globulin, thyroxine-binding pre albumin and albumin in plasma.

Caregaro, et al. (1998) reported that low T4 variants of sick euthyroid syndrome has been associated with decreased short- and long-term survival of patients with liver cirrhosis.^[29] Other studies, indicated that low serum T3 is a good index of disease severity in cirrhosis.^[17,13] Reverse T3 was accompanied with development of hepatocellular carcinoma in HCV cirrhosis.^[11] A retrospective study suggested liver function tests tend to be better in hypothyroid state.^[30] The complications found in our study correlates with and is well supported by the other studies like that in P Puneekar et al,^[20] (2018) and Kharb S et al,^[25] (2015).

CONCLUSION

Thyroid abnormalities are not uncommon in patients with cirrhosis. Hypothyroidism is the most common abnormality that was detected. Low T3 and FT4 level is significantly associated with more severe liver injury in patients with cirrhosis of liver. Low levels of T3 inversely correlated with the severity of liver disease in the form of CTP or MELD. Level of FT4 decreases as CTP Class increases. Level of TSH increases with MELD score. Therefore, thyroid levels in cirrhotic patients may be used as a prognostic marker.

So, thyroid function test should be carried out in all cirrhotic patients to assess the severity and prognostication of such patients.

Acknowledgements: Authors would like to acknowledge the patients who participated in this research study.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: The study was approved by the institutional ethics committee

REFERENCES

1. Global, regional, and national age-sex-specific mortality for 282 causes of death in 195 countries and territories, 1980–2017: a systematic analysis for the Global Burden of Disease Study 2017. *Lancet*. 2018; 392: 1736–1788.
2. Anthony PP, Ishak KG, Nayak NC, Poulsen HE, Scheuer PJ, Sobin LH. The morphology of cirrhosis. Recommendations on definition, nomenclature, and classification by a working group sponsored by the World Health Organization. *J Clin Pathol* 1978; 31: 395–414.
3. Fleming KM, Aithal GP, Card TR, West J. The rate of decompensation and clinical progression of disease in people with cirrhosis: a cohort study. *Aliment Pharmacol Ther* 2010; 32: 1343–50.
4. Moreau R, Jalan R, Gines P, et al. Acute-on-chronic liver failure is a distinct syndrome that develops in patients with acute decompensation of cirrhosis. *Gastroenterology* 2013; 144: 1426–37.e1–9.
5. D'Amico G, Garcia-Tsao G, Pagliaro L. Natural history and prognostic indicators of survival in cirrhosis: a systematic review of 118 studies. *J Hepatol* 2006; 44: 217–31.
6. Fleming KM, Aithal GP, Card TR, West J. All-cause mortality in people with cirrhosis compared with the general population: a population-based cohort study. *Liver Int* 2012; 32: 79–84.
7. Ge PS, Runyon BA. Treatment of patients with cirrhosis. *N Engl J Med* 2016; 375: 767–77.
8. Rockey DC, Caldwell SH, Goodman ZD et al., American Association for the Study of Liver D. Liver biopsy. *Hepatology* 2009; 49: 1017–1044.
9. McCormick PA. Sherlock's Diseases of the Liver and Biliary System Textbook. Hepatic Cirrhosis. In: Dooley JS, Lok AS, Burroughs AK, Heathcote EJ, editors. 12th ed. Ch. 7. UK: Wiley-Blackwell, A John Wiley and Sons, Ltd; 2011. pp. 103–8.
10. Jameson JL, Mandel SJ, Weetman AP. Harrison's Principles of Internal Medicine Textbook. Disorders of the Thyroid gland. In: Kasper DL, Hauser SL, Jameson JL, Fauci AS, Longo DL, Loscalzo J, editors. 19th ed. Ch. 405. New York: McGraw-Hill Education; 2015. pp. 2283–2308.
11. Sorvillo F, Mazziotti G, Carbone A, Morisco F, Cioffi M, Rotondi M, et al. Increased serum reverse triiodothyronine levels at diagnosis of hepatocellular carcinoma in patients with compensated HCV-related liver cirrhosis. *Clin Endocrinol (Oxf)* 2003;58:207–12.

12. Kharb S, Garg MK, Puri P, Brar KS, Pandit A, Srivastava S, et al. Assessment of thyroid and gonadal function in liver diseases. *Indian J Endocrinol Metab.* 2015;19:89–94.
13. Kayacetin E, Kisakol G, Kaya A. Low serum total thyroxine and free triiodothyronine in patients with hepatic encephalopathy due to non-alcoholic cirrhosis. *Swiss Med Wkly.* 2003;133:210–3.
14. Malik R, Hodgson H. The relationship between the thyroid gland and the liver. *QJM.* 2002;95:559–69.
15. Hasselbalch HC, Bech K, Eskildsen PC. Serum prolactin and thyrotropin responses to thyrotropin-releasing hormone in men with alcoholic cirrhosis. *Acta Med Scand.* 1981;209:37–40.
16. D'Azzò G, Pinzello GB, Pace F, Garofalo P, Craxi A, Janni A, et al. The prognostic value of thyroid function tests in predominantly non-alcoholic cirrhotic patients: A prospective investigation. *J Endocrinol Invest.* 1985;8:331–6.
17. Taghavi SA, Eshraghian A, Hamidpour L, Moshfe MJ. Endoscopic cyanoacrylate injection for the treatment of bleeding gastric varices: the first Iranian series. *Arch Iran Med.* 2012; 15: 157 – 161.
18. Mansour-Ghnaei F, Mehrdad M, Mortazavi S, Joukar F, Khak M, Atrkar-Roushan [1]Z . Decreased serum total T3 in hepatitis B and C related cirrhosis by severity of liver damage. *Annals of Hepatology.* 2012;11(5):667-671
19. Verma S kumar, Kumar V, tiwari P, Joge NKP, Misra R. Thyroid Profile in Patients of Cirrhosis of Liver: A Cross-sectional Study. *J Clin Diagn Res .* 2017; Vol-11(12): OC06-OC09
20. Puneekar P, Sharma AK, Jain A. A study of thyroid dysfunction in cirrhosis of liver and correlation with severity of liver disease. *Indian J EndocrMetab*2018;22:645-50
21. Samarthana V, Mamatha BP. Study of Thyroid Function Tests Incirrhosis of Liver And Correlation of Thyroid Function Tests Levels With Severity of Liver Dysfunction. *J Endo Metabol Res.* 2020;1(2):1-13
22. Ashish Kumar, Vivek Ahuja, Inderjitkaur, VandnaPandov, Amritpal Singh and RPS Sibia; Prevalence of thyroid dysfunction in patients of cirrhosis of liver and its correlation with severity of cirrhosis; *Int. J. Adv. Res.*2020 8(04), 91-95
23. Kalmani VM, Madhuvan HS, Ravishankar SN, et al. A cross-sectional study of thyroid dysfunction in patients suffering from liver cirrhosis in a tertiary care hospital in Bengaluru, India. *J Evid Based Med Health* 2019;8(23):1904-1908
24. Joeimon JL, Mohanraj K, Karthikeyan R, et al. Thyroid dysfunction in patients with liver cirrhosis. *IOSR J Dent Med Sci* 2017;16(4):18-22.
25. Kharb S, Garg MK, Puri P, et al. Assessment of thyroid and gonadal function in liver diseases. *Indian J Endocrinol Metab* 2015;19(1):89-94.
26. Buden RP. Thyroid Dysfunction in Patients with Liver Disease. *Acad. J Med.* 2019;2(2):138-40.
27. M Borzio, R Caldara, F Borzio, V Piepoli, P Rampini, and C Ferrari. Thyroid function tests in chronic liver disease: evidence for multiple abnormalities despite clinical euthyroidism. *Gut,* 1983, 24, 631-636
28. Mobin A, Shaikh H, Haroon H, et al. Decompensated cirrhosis: Thyroid hormone levels in patients. *The Prof Med J* 2016;23(1):34-38
29. Caregaro L, Alberino F, Amodio P, Merkel C, Angeli P, PlebaniM,et al. Nutritional and prognostic significance of serum hypothyroxinemia in hospitalized patients with liver cirrhosis. *J Hepatol.* 1998;28: 115 – 121
30. Oren R, Brill S, Dotan I, Halpern Z. Liver function in cirrhotic patientsin the euthyroid versus the hypothyroid state. *J Clin Gastroenterol.*1998; 27: 339 – 341.