Assessment of Thyroid Gland Status in Children and Adolescents with Chronic Liver Disease

Original Article

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ABSTRACT

Background: Thyroid hormone metabolism may be impaired in chronic liver diseases. Non-thyroidal illness (NTIS) or subclinical hypothyroidism (SH) or even overt hypothyroidism may occur.

Aim of the Work: To assess thyroid gland status in children and adolescents with chronic liver diseases (CLD).

Methods: This cross- sectional controlled study was conducted on 40 children with chronic liver diseases recruited from Pediatric Hepatology Clinic, Children's Hospital, Ain-Shams University and compared to 40 age- and sex- matched healthy controls. History taking and physical examination were performed. Assessment of severity using modified Child-Pugh score was done. Laboratory investigations in the form of liver functions and thyroid profiles were done.

Results: Twenty males (50%) and twenty females (50%) were included in the study. 67.5% of patients with CLD had non-cholestatic liver diseases. Biliary atresia and congenital hepatic fibrosis were the most frequent causes of CLD. About 75 % and 22.5 % of the patients had Child-Pugh A and B cirrhosis respectively. Normal thyroid function was detected in 95% of cases, while 5% had subclinical hypothyroidism despite most of our cases were euthyroid, they had significantly lower T3 ($p< 0.001$) and higher TSH levels ($p < 0.017$). TSH was significantly higher in group of patients with Child-Pugh B/C class. Free T3 had significant negative correlation with total bilirubin. TSH had significant positive correlations with total and direct bilirubin.

Conclusion: Thyroid dysfunction might occur in children with CLDs. Children with CLD demonstrated an increase in TSH levels. Thus, screening of thyroid function is recommended when investigating patients with CLD.

Key Words: Children, liver disease, thyroid gland.

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INTRODUCTION

Liver plays an important role in metabolism of thyroid hormones like conjugation, excretion, peripheral deiodination and synthesis of thyroid-binding globulin[1,2]. So thyroid dysfunction has been reported in various spectra of liver diseases and is associated with the severity of liver disease[3,4].

On the other hand, thyroid hormones play a fundamental role in lipid metabolism and mitochondrial function in the liver, overt or subclinical hypothyroidism has been reported to affect the management and prognosis of liver disease[5]. Thyroid gland impairment could represent with biochemical abnormalities due to liver dysfunction and pathological changes associated with end organ damage. Acute liver failure is associated with increased circulating endotoxins and proinflammatory mediators, which is quite similar to clinical state of sepsis and results in dysfunction of endocrinal glands like non-thyroidal illness syndrome (NTIS)[6].

Thyroid dysfunction has been reported in 5% to 20% of patients with chronic active hepatitis, a frequency which is higher than reported for general population[7]. Most of

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these patients have various degree of hypothyroidism. Isolated low free triiodothyronine (fT3) was seen in significant number of patients. Additionally, the grade of hypothyroidism was found to be significantly linked to deterioration in liver functions and becoming more evident with late-stage liver failure and cirrhosis[8].

In stable cirrhosis, a state of hypothyroidism has been shown to correlate with slow progression of stages of cirrhosis[9]. Child-Pugh Score categories were found to be related to the mean score of fT3 and the mean score of free tetraiodothyronine (fT4)[8].

In patients with end stage liver disease with low levels of fT3 and fT4 subjected to liver transplantation, the level of these hormones normalized after transplantation[10,11]. So, all the liver cirrhotic patients should be evaluated for thyroid dysfunction for early diagnosis and management[8]. Early identification of at-risk patients is important, since treatment of the hypothyroidism may reduce the risk of non-cholestatic chronic liver disease complications[12].

AIM OF THE WORK

To investigate thyroid gland status in children and adolescents with chronic liver diseases.

SUBJECTS AND METHODS

This cross-sectional controlled study was conducted at Pediatric Hepatology Clinic, Ain-Shams University Children's Hospital during the period from June 2022 till June 2023. Forty patients with chronic liver diseases (CLD) were recruited from Hepatology Clinic and forty age- and sex- matched children and adolescents recruited from Outpatient Clinic (healthy siblings of patients in ER or coming to outpatient clinic due to minor complaints) served as a control group.

Patients aged from one year to 14 years with chronic liver diseases were included in the study. While children and adolescents with pre-existing thyroid disease were excluded from the study. Children and adolescents on chronic medications that might affect thyroid function such as (Amiodarone), the antimalarial drug (mefloquine), the anti-epileptic drug (carbamazepine), chemotherapy, and radiotherapy have all been associated with both thyroid and liver toxicity also they were excluded from the study[13].

All studied cases were subjected to complete history taking (with special emphasis on personal history, underlying liver disease, symptoms of hypothyroidism & hyperthyroidism, any comorbidity as short stature and delayed puberty), physical examinations (with special emphasis on anthropometric parameters, tanner staging, assessment of liver condition, assessment of thyroid gland), laboratory investigations in the form of thyroid function test including TSH, fT3, and fT4 for both groups, and thyroid gland ultrasound whenever deemed essential.

Thyroid gland ultrasound wasn't performed for any of our patients because none of them had abnormal thyroid enlargement, any palpable nodule or any abnormal thyroid vascularity.

Study protocol was submitted for approval by the institutional review board (IRB) of Faculty of Medicine, Ain Shams University, Children's Hospital. A written informed consent was obtained from the parents or guardians of the patients or controls before inclusion in the study.

ETHICAL CONSIDERATION

Approval of the Research Ethic Committee faculty of Medicine, Ain Shams University was obtained before start of the study no FWA 000017585 on 11/1/2022. Confidentiality and personal privacy were respected in all the levels of the study. Collected data was not to be used for any other purpose.

Biochemical assessment

Under complete aseptic conditions 2 ml venous blood were withdrawn into a plan tube, left to clot, centrifuged and stored at -70°C for determination of thyroid functions (FT3, FT4, TSH). They were analyzed by the 'ECLIA' chemiluminescence method on a Roche Elecsys E170 (Roche Diagnostics, Indianapolis, IN, Germany). TSH, fT4 and fT3 concentrations of the patients included in the study were evaluated by reference to the age range of this kit[14]. CBC, liver function tests results were obtained from patients' files.

Statistical methods: The collected data were coded, tabulated, and statistically analyzed using IBM SPSS statistics (Statistical Package for Social Sciences)

software version 28.0, IBM Corp., Chicago, USA, 2021. Quantitative data tested for normality using Kolmogorov-Smirnov test, then if normally distributed described as mean± SD (standard deviation) as well as minimum and maximum of the range, and then compared using independent t-test in addition to Person correlation test, and if not normally distributed described as Median (1^{st}–3^{rd} Interquartiles) as well as minimum and maximum of the range and then compared using Mann Whitney test in addition to Spearman correlation test. Qualitative data described as number and percentage and then compared using Chi square test as well as Fisher's exact test. Linear regression was done for factors affecting fT3, f T4 and TSH. The level of significance was taken at *p-value* ≤0.050 was significant, otherwise was non-significant.

RESULTS

This cross-sectional controlled study included 40 subjects; 20 males (50%) and 20 females (50%) aged from 1 year to 14 years old. The median age at diagnosis of CLD group was 82 months.

Demographic data of studied cases is shown in (Table 1). 75% of cases were prepubertal and 25% entered puberty with respect to Tanner staging. 30 (75% out of 40 cases were of middle socioeconomic class, 13 cases (32.5%) were of low socioeconomic class, whereas only 3 cases (7.5%) were of high socioeconomic class.

Table 1: Demographic characteristics, anthropometric measures and Tanner staging among CLD patients group.

Variables		Median (1^{st}–3^{rd} IQ)	Range
Age (months)		82.0 (38.5–136.8)	11.0–175.0
		n	%
Sex	Male	20	50.0%
	Female	20	50.0%
Positive consanguinity		13	32.5%
Socioeconomic class (n, %)	Low	7	17.5%
	Middle	30	75.0%
	High	3	7.5%
Variables		Mean± SD	Range
Weight for ht.(SDS)		-1.3±2.3	-8.7–1.7
Height for age(SDS)		-1.3±1.8	-5.7–3.6
BMI (SDS)		-0.4±2.4	-7.8–4.1
		n	%
Tanner stage	T1	30	75.0%
	T2	5	12.5%
	T3	5	12.5%

IQ: Interquartiles. BMI: Body mass index.

The most common etiology for CLD was Biliary atresia compromised 10 cases out of 40 cases (25%), followed by Congenital hepatic fibrosis 7 cases (17.5%) and Autoimmune hepatitis 6 cases (15%) as shown in (Table 2).

Table 2: Categorization and etiological classification of patients included in CLD group.

Variables	Number (total=40)	%
Diagnosis		
Biliary atresia	10	25.0%
Congenital hepatic fibrosis	7	17.5%
Autoimmune hepatitis	6	15.0%
Gaucher syndrome	4	10.0%
Chronic hepatitis C	3	7.5%
Allagille syndrome	2	5.0%
Budd chiari syndrome	2	5.0%
Glycogen storage liver disease	2	5.0%
Tyrosinemia	2	5.0%
Hyperphenylalaninemia	1	2.5%
Progressive familial intra hepatic cholestasis	1	2.5%

Constipation and dry skin were the most common manifestations of hypothyroidism among CLD patients with percentage 37.5% (15 cases) & 25.0% (10 cases) respectively. Cold intolerance manifested in 12.5% (5 cases). Symptoms of hyperthyroidism were negative in all CLD patients.

Three quarters of patients studied 30 cases (75.0%) were Child-Pugh A as shown in (Table 3).

Table 3: Child Pugh classification of CLD patients group.

		Number (total=40)	%
Child-Pugh	A	30	75.0%
	B	9	22.5%
	C	1	2.5%

Evidence of anemia was present in 27 cases (67.5%) and thrombocytopenia in 29 cases (72.5%) mostly denoting hypersplenism. Patients showing hyperbilirubinemia were 24 cases (60%), about 26 cases (65%) had prolonged INR and 21 cases (52%) had elevated transaminases. 22 cases (55%) had elevated GGT.

CLD group had significantly lower T3 and significantly higher TSH compared to control group. However, these levels were still within normal range in both CLD patients and controls as shown in (Table 4).

Table 4: Comparison between CLD and control groups regarding thyroid function profile.

Variables		CLD group (Total=40)	Control group (Total=40)	*p-value*
FreeT3 (pg/mL)	Mean± SD	2.9±0.5	3.3±0.5	^<0.001*
	Range	1.9–4.0	2.1–4.2	
FreeT4 (ng/mL)	Mean± SD	1.5±0.2	1.4±0.2	^0.225
	Range	1.0–2.0	1.1–2.0	
TSH (mIu/L)	Mean± SD	3.2±1.2	2.6±1.1	^0.017*
	Range	1.8–8.2	0.9–4.5	
High TSH		2 (5.0%)	0 (0.0%)	§0.494

NA: Not applicable. ^Independent t-test. §Fisher's Exact.

FT3 was significantly lower in child B/C cases and TSH was significantly higher in the same group (*p- value* 0.002, 0.029 respectively) but both were still in the normal range.

In this study, high TSH was reported in only 2 patients (5%). However, free T3 and T4 were normal in all studied CLD patients.

Free T3 had significant negative correlation with both age at diagnosis and total bilirubin (*p-value*= 0.016, 0.030 respectively). TSH had a significant negative correlation with albumin and a significant positive correlations with both total bilirubin and direct bilirubin. Free T4 showed no significant correlation with any of the variables as shown in (Table 5).

Table 5: Correlations of thyroid function profile with clinical characteristics and laboratory investigations among chronic liver disease (CLD) group.

Variables	FreeT3		FreeT4		TSH	
	r	*p-value*	r	*p-value*	r	*p-value*
⌂Age at diagnosis	-0.378	0.016*	-0.250	0.120	0.245	0.128
⌂Duration of illness	-0.168	0.299	0.046	0.780	0.237	0.141
^Hemoglobin	0.076	0.640	0.040	0.807	-0.108	0.507
^Platelets	-0.041	0.802	0.049	0.765	0.042	0.798
^Albumin	0.164	0.311	-0.051	0.755	-0.442	0.004*
^Total protein	0.064	0.695	0.146	0.368	0.005	0.977
^Total bilirubin	-0.343	0.030*	-0.147	0.364	0.427	0.006*
^Direct bilirubin	-0.300	0.060	-0.128	0.433	0.574	<0.001*
^ALT	0.124	0.447	0.051	0.756	-0.143	0.379
^AST	0.125	0.444	0.142	0.382	-0.031	0.849
^GGT	0.089	0.585	0.038	0.815	0.159	0.327
^Alkaline phosphatase	0.098	0.547	0.133	0.415	0.164	0.311
^PT	-0.202	0.211	-0.063	0.701	0.235	0.145
^PTT	0.072	0.658	-0.002	0.991	-0.129	0.426
^INR	-0.293	0.067	-0.203	0.209	0.005	0.977

Total=40. ⌂Spearman correlation. ^Pearson correlation.

Total protein and male sex were significant independent factors that increased T3, while Child B was a significant independent factor that decreased T3. Total protein and male sex were significant independent factors that increased T4. Total protein and Child B were significant independent factors that increased TSH as shown in (Table 6).

Table 6: Linear regression analysis for factors affecting T3, T4 and TSH.

Factors	β	SE	*p-value*	95% CI	R^2
Free T3					
Total protein	0.387	0.043	<0.001*	0.300–0.473	0.967
Male sex	0.532	0.154	0.001*	0.220–0.845	
Child B/C	-0.667	0.204	0.002*	-1.079–-0.254	
Free T4					
Total protein	0.196	0.020	<0.001*	0.155–0.237	0.971
Male sex	0.240	0.073	0.002*	0.093–0.388	
TSH					
Total protein	0.488	0.037	<0.001*	0.412–0.563	0.893
Child B/C	1.581	0.427	0.001*	0.717–2.445	

β: Regression coefficient, SE: Standard error, CI: Confidence interval, *significant, R^2: Coefficient of determination.

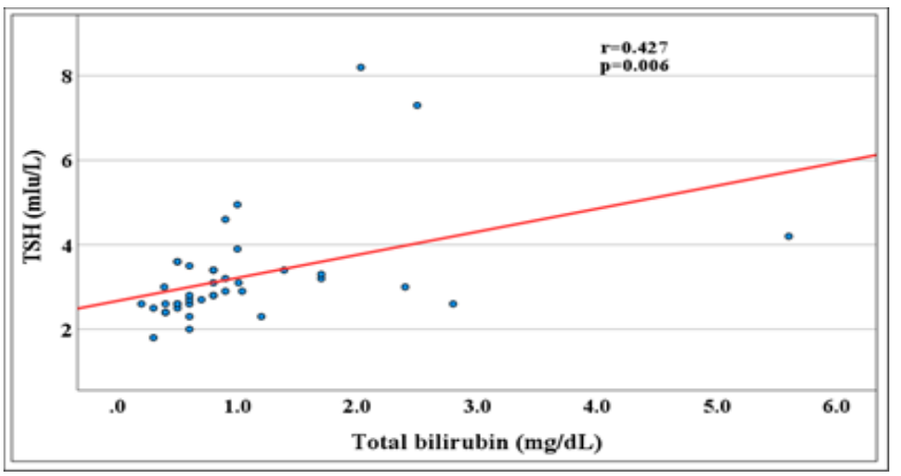

Fig. 1: Correlation between T3 and total bilirubin among CLD group

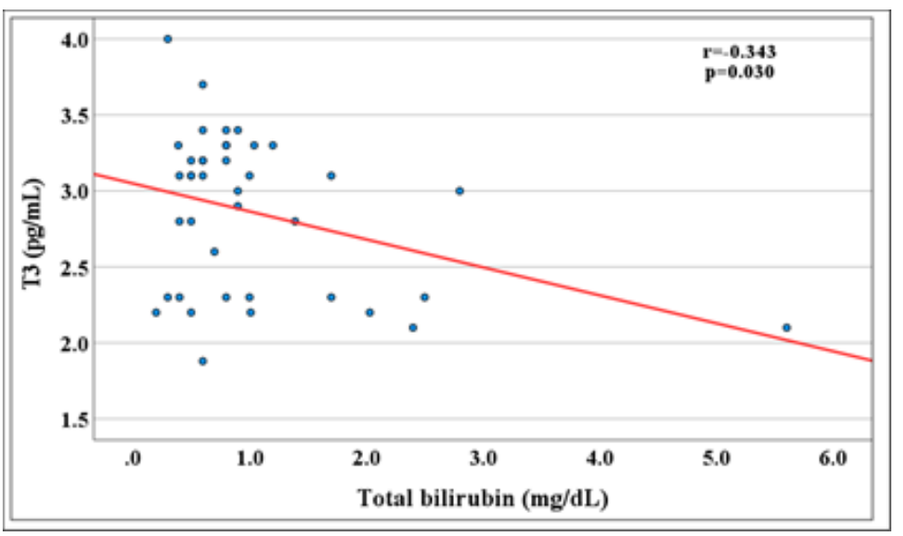

Fig. 2: Correlation between TSH and total bilirubin among CLD group.

DISCUSSION

The liver has an important role in metabolism of thyroid hormones. Thyroid function disturbance could be associated with chronic liver disease. To the best of our knowledge, there is a paucity of studies in literature evaluating the thyroid functions in chronic liver diseased children, and that represents a strength point of our study.

We aimed in our study to investigate thyroid gland status in children and adolescents with CLD. Forty patients with CLD were enrolled in the study. The age of patients in this study ranged from 1 year till 14 years old making two equal sex groups with median age of 6.83 years. The median age at diagnosis of CLD was 6 years old with a mean of 6.5 ± 3.2 years.

Most of our cases were from the middle socioeconomic class (75%) followed by low socioeconomic class (17.5%) with no significant difference between cases and controls in socioeconomic class.

Our study reported that biliary atresia and congenital hepatic fibrosis were the most frequent causes of CLD (25% and 17.5% respectively). Autoimmune hepatitis and viral hepatitis represented together (22.5%). Parental consanguinity was reported in (32.5%) of CLD cases.

On the other side, ***Behairy et al.***[15] studied CLD in children in Menofia and revealed that metabolic liver diseases (Wilson, Glycogen storage disease, α1 antitrypsin deficiency, galactosemia) were the most frequent causes of CLD representing 29.13% followed by viral hepatitis (hepatitis C and B) which represented 23.30%. This could be explained by higher consanguinity rates and low sanitary level in the countryside in Egypt compared to Cairo.

According to anthropometric measurements, the present study showed that CLD cases group had significantly lower weight, height and BMI SDS compared to control group. The mean BMI (SDS) was -0.4±2.4 in CLD group. Nearly quarter of studied cases had weight and height SDS less than -2 (30%, 21.5% respectively). This could be attributed to loss of appetite, frequent hospital admissions and chronic illness in CLD group[15].

Our results could be also compared to those obtained by ***Ön et al.***[7] who found that mean BMI SDS in CLD group was -0.80±1.79.

Similar results were previously obtained by ***Behairy et al.***[15] in another centre in Egypt (Menofia) who aimed to assess nutritional status in CLD patients through anthropometric measurements.

Seventy five % and 23% of the studied patients had Child-Pugh A and Child-Pugh B respectively. While only one patient had Child-Pugh C cirrhosis.

On the other hand, in a similar study in Alexandria University in Egypt, they found Class A Child Pugh represented 83.3% of CLD patients, and Class B were about 16.7% of CLD patients. There were no cases with Class C[16].

Subclinical hypothyroidism is defined as a serum TSH concentration above the reference range with normal serum fT4 and fT3 levels[17,18]. Regarding the thyroid profile, our study results showed that normal thyroid function was detected in 95% of cases, whereas 5% had subclinical hypothyroidism (the 2 cases were; GSD and Gaucher disease).

The first case with subclinical hypothyroidism was a 4 years old female patient diagnosed as GSD at the age of 18 months with hepatomegaly and classified as Child-Pugh class A that is not in favor with NTIS. By reviewing the literature thyroid function affection comes in turn because of the liver affection not as a part of original disease[19].

The second case had Gaucher disease was a 9 years old male, diagnosed at the age of 8 months with hepatomegaly and bleeding liability. The patient was Child-Pugh class A, this excludes Non thyroidal illness syndrome (NTIS). Also by reviewing the literature thyroid affection comes secondary to liver affection not as a part of original disease[20, 21].

Similar results were obtained by ***Ön et al.***[7] who revealed that 89.7% of CLD patients had normal thyroid function test, 6.5% had subclinical hypothyroidism.

But higher percentages of subclinical hypothyroidism were obtained by Kim[22]. Nearly a quarter of the studied CLD pediatric patients had subclinical hypothyroidism.

Interestingly, despite most of our cases were euthyroid, they had significantly lower T3 and higher TSH levels compared to controls with normal T4 levels.

Other studies done by ***Mobin et al.***[23], ***El-Feki et al.***[24], ***Punekar et al.***[25] and ***Bebars et al.***[26] reported that all decompensated cirrhotic patients had low serum T3 levels, serum T4 levels, and raised TSH levels.

Studies by ***Torun et al.***[27], ***Vincken et al.***[28], ***Punekar et al.***[25], ***Ön et al.***[7] and ***Raj et al.***[29] revealed a positive and direct correlation associated between increasing TSH with severity of CLD as measured by Child-Pugh score, they stated that in all cirrhotic patients, fT3 and fT4 were negatively correlated, but TSH level was positively correlated with serum bilirubin and liver function tests.

These results are comparable to our results as regards FT3 and TSH. TSH was significantly higher in Child Pugh B and C compared to Child Pugh A group. FT3 was significantly lower in Child Pugh B and C cases compared to Child Pugh A cases.

The present study showed that Child Pugh score is a significant independent factor that affects TSH. Also, the previous studies revealed positive correlations that suggest that the Child-Pugh score can be used as a prognostic indicator in patients with CLD affecting thyroid function in children and is correlated with the disease severity[29].

In a study by ***Mahfouz et al.***[16], they reported no significant correlation between thyroid dysfunction and modified Child Pugh score. As they reported no cases with Child Pugh class C between their CLD pediatric patients.

Also, a study of ***Kharb et al.***[30] reported a decrease in the levels of TSH. Other comparative studies by ***Vincken et al.***[28] and ***Punekar et al.***[25] reported significantly lower levels of both FT3 and FT4 alongside normal TSH values in cirrhotic patients compared to healthy controls. The observed previous patterns might be associated with increase in inflammatory cytokines that negatively affect hypothalamo-thyroid axis which may suggest a pattern consistent with relative and functional central hypothyroidism, commonly observed in Non-thyroidal illness syndrome (NTIS)[31].

Other contributing factors that explain lower T3 in pediatric patients with CLD include alteration in plasma level of thyroid binding proteins, altered binding of T4 and T3 to their carrier protein, impaired hepatic clearance of reverse T3 and reduced extra thyroidal conversion of T4 to T3. In cirrhotic patients, because of extensive hepatic inflammation and fibrosis, there is inhibition of Type 1 deiodinase enzymes that lead to decreased conversion of T4 to T3[26].

Many studies state that the incidence of NTIS is significantly higher in patients with CLD of various causes, including cirrhosis and end-stage liver disease, and that alteration of thyroid hormone concentration is associated with the severity of liver disease[15].

Kharb et al.[30] reported that 18.7% with acute and CLD had thyroid dysfunction and 8% of these patients were reported to have NTIS. Also, ***Ön et al.***[7] reported 3.7% of CLD patients had NTIS.

This difference between the aforementioned studies and our study might be due to difference in severity of liver disease and regional variation of thyroid disorders.

Free T3 had a significant negative correlation and TSH had significant positive correlations with total bilirubin. Total protein was significant independent factor that increased T3, T4 and TSH. While Child B was a significant independent factor that decreased T3 and increased TSH. Free T3 had significant negative correlation with both age at diagnosis and total bilirubin.

Furthermore, in the study by Kim[22] they reported significant positive associations between TSH, AST, and total bilirubin in patients with CLD having subclinical hypothyroidism. Patients with subclinical hypothyroidism had more severe abnormalities in liver function tests than those without this condition. This finding suggests the possibility of more liver cell damage in patients with excessive TSH levels.

Ön et al.[7] revealed that the correlation analysis between thyroid function tests and liver function tests revealed a negative correlation between fT3 and direct bilirubin. It has been reported that serum fT3 concentrations are associated with the severity of liver function, based on liver function test results.

Vincken et al.[28] showed a statistically significant positive correlation between fT3 and ALT and albumin and a positive correlation between increasing TSH and severity of liver cirrhosis as measured by Child-Pugh score, whereas a negative and inverse correlation was observed between

decreasing fT3 and fT4 levels and the severity of liver cirrhosis as measured by Child-Pugh score. This suggests that the Child-Pugh score can be used as a prognostic indicator in cirrhotic patients. Consequently, fT3 correlates inversely with severity of liver disease in patients with liver cirrhosis.

Another study was done by ***El-Kabbany et al.***[32] and ***Mahfouz et al.***[16], It was found that serum albumin level was significantly lower with subclinical hypothyroidism (SH) subgroup. They found a positive correlation between FT3 and each of serum albumin and total proteins and explained that poor nutritional factors were implicated in the low T3 patients with CLD.

On the other side, ***Khan et al.***[33] and ***Bebars et al.***[26], illustrated that there was no significant correlation between thyroid function tests and liver enzymes in patients with CLD.

In our study, none of the patients has hyperthyroid clinical and laboratory evidence. Also, no other papers found a significant hyperthyroidism except as a part of autoimmune disease.

Based on these observations, it is reasonable to suggest that thyroid function tests should be regularly checked in patients with liver cirrhosis and prompt treatment initiated in case of overt or subclinical hypothyroidism (elevated TSH and normal-to-low FT4 and FT3), whereas it is not indicated to treat isolated low FT3[4].

The strength point of this study is that it shows how crucial for clinicians to be aware of the connection between hypothyroidism and abnormal liver function indicators. This knowledge helps them in assessing thyroid function when investigating patients with chronic liver disease.

As evident from the current study, the prevalence of subclinical hypothyroidism was around 5% of children and adolescents with chronic liver diseases. Therefore, thyroid function testing is suggested in these cases at diagnosis and during follow-up. Total protein and Child B score were significant independent factors that were associated with increased TSH.

The limitations of the study include relatively small sample size, not being a multi-centric study and this represents a significant risk of publication bias. Another limitation is that laboratory evaluation for dyslipidemia and thyroid antibodies were not performed. So, we could not evaluate their association with subclinical hypothyroidism in patients with liver disease.

Therefore, clinicians might frequently consider thyroid function tests in patients with liver disease, especially younger patients and those with elevated total bilirubin and direct bilirubin levels.

CONCLUSION

As evident from the current study, the prevalence of subclinical hypothyroidism in this study is around 5% of children and adolescents with chronic liver diseases which signifies the importance of screening for thyroid dysfunctions in CLD patients. Follow-up of the patients is mandatory to document changes in thyroid hormone levels in CLD patients over time. Further studies using larger cohort should be done to confirm our results.

CONFLICT OF INTEREST

- There has been no funding for this research, and there is no competing personal financial interests in relation to the work described.
- The person who wrote the first draft of the manuscript is: Dr Nadin Nabil Toaima.
- Each author listed in the manuscript has seen and approved the submission of this version of the manuscript and takes full responsibility for the manuscript.

AUTHORS CONTRIBUTIONS

1. Dr. Nadin Nabil toaima: She shared in putting the concept of the study. She shared in interpretation of data and final approval of the version to be published. She wrote the first draft and revised the final script for publication.
2. Eman M Mohsen: She is the corresponding author. She did the clinical work, recruited the case and shared in data collection and analysis, interpretation of results and she shared in literature search.

3. Dr. Iman M Talaat: She shared in putting the study design, and in recruitment of cases analysis of data and interpretation of results. She revised the results.

4. Dr Menat Alla A Shaaban: She revised the results and shared in data collection and analysis, interpretation of results.

5. Dr. Rana AA Mahmoud: She shared in study design, analysis of data, interpretation of results and revised the results.

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تقييم حالة الغدة الدرقية عند الاطفال و المراهقين المصابين بامراض الكبد المزمنة

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الخلفية: قد يضعف استقلاب هرمون الغدة الدرقية في الاطفال المصابين بأمراض الكبد المزمنة. قد يحدث ذلك متلازمة المرض غير الدرقي أو قصور الغدة الدرقية دون السريري أو حتى قصور الغدة الدرقية البين.

الأهداف: التحقيق في حالة الغدة الدرقية لدى الأطفال والمراهقين المصابين بأمراض الكبد المزمنة.

الطرق: أجريت الدراسة المستعرضة دراسة الحالات والشواهد هذه على ٤٠ مريضًا مصابًا بأمراض الكبد المزمنة تم تجميعهم من عيادة أمراض الكبد للأطفال، مستشفى الأطفال، جامعة عين شمس مقارنة بـ ٤٠ طفلًا ومراهقًا بصحة جيدة مطابقين للعمر والجنس. تم إجراء الفحص البدني الكامل، تم تطبيق مقياس درجة شدة حالة الكبد Child-Pugh، وتم إجراء اختبار مستوي الغدة الدرقية، وفحص وظائف الكبد في الدم.

النتائج: تمت الدراسة علي ٢٠ ولد (٥٠٪) و ٢٠ بنت (٥٠٪). أفادت دراستنا أن ٦٧,٥٪ من مرضى الكبد المزمن كانوا مصابين بأمراض الكبد الصفراوي غير الركودي. كان لدى حوالي ٧٥٪ و ٢٣٪ من المرضى تليف الكبد من النوع A و B على التوالي. تم الكشف عن وظيفة الغدة الدرقية الطبيعية في ٩٥٪ من الحالات، بينما كان لدى ٥٪ قصور الغدة الدرقية دون السريري على الرغم من أن معظم حالاتنا كانت وظائف الغدة الدرقية سليمة، إلا أن لديهم مستويات منخفضة بشكل ملحوظ من هرمون ثلاثي يودوثيرونين الحر(FT3) ومستويات أعلى من الهرمون المحفز للغدة الدرقية (TSH). كانت فئة Child-Pugh عاملاً بارزا مهمًا أدى إلى انخفاض هرمون الغدة الدرقية الحر هرمون ثلاثي يودوثيرونين الحر (FT3) وزيادة الهرمون المحفز للغدة الدرقية (TSH) وقد كان هرمون الغدة الدرقية المحفز (TSH) أعلي بشكل كبير في مجموعة المرضي الذين يعانون من فئة Child-Pugh B/C. كان لهرمون T3 الحر ارتباط سلبي بمستوى البيليروبين الكلي. كان للهرمون المحفز للغدة الدرقية (TSH) ارتباط إيجابي كبير بكل من البيليروبين المباشر والبيليروبين الكلي.

الخلاصة: كما يتضح من الدراسة الحالية، يُظهر الأطفال المصابون بمرض الكبد المزمن زيادة في مستويات هرمون الغدة الدرقية. لذلك يوصى بفحص وظائف الغدة الدرقية عند فحص المرضى المصابين بأمراض الكبد المزمنة.