Evaluation of Liver Function Tests in Patients with Hypothyroidism in a Tertiary Care Hospital

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ABSTRACT

Background

Thyroid hormones are essential for normal organ growth and metabolic regulation, including the basal metabolic rate. Hypo functioning of thyroid gland often results in abnormal liver function tests, even without overt liver disease. This study aimed to assess these hepatic alterations by comparing liver function parameters between hypothyroidism and euthyroidism attending a tertiary care hospital.

Methods

An analytical cross-sectional study was conducted in a tertiary care hospital from 1st August to 30th November 2025 with consecutive sampling technique. Ethical clearance was taken from institutional review committee (IRC reference No. 25052025/16). 145 participants who were diagnosed as hypothyroidism on the basis of Thyroid function test (TFT) were enrolled in the study who were further grouped as sub-clinical and overt hypothyroidism. Liver function test (LFT) and TFT were assessed through an automated analyzer and Chemiluminescence Immune Assays (CLIA) methods respectively.

Results

Among the hypothyroid subjects, majority of the participants belong to subclinical hypothyroidism 109 (75.2%), whereas 36 (24.8%) were grouped as overt hypothyroidism. Serum bilirubin was significantly higher in overt hypothyroidism. Serum aminotransferase (AST and ALT) were higher but total protein was lower in hypothyroidism. In this study 19(13.1%) and 13(9%) of the hypothyroidism participants had elevated aminotransferase activity.

Conclusion

This study revealed a significant rise in serum ALT and AST in hypothyroid subjects compared to that in controls. Furthermore, serum albumin and Alkaline Phosphatase (ALP) were positively significantly correlated with Thyroid Stimulating Hormone (TSH).

Keywords

Liver Function Tests, Overt hypothyroidism, Subclinical hypothyroidism, Thyroid Function Tests

Introduction

Hypothyroidism is a common endocrine disorder due to hypo functioning of the thyroid gland,[1] and the prevalence of which is more common in women across the all age group.[2] Thyroid hormones include triiodothyronine (T3) and thyroxine (T4) which are produced in response to thyroid-stimulating hormone (TSH) from anterior pituitary gland which in turn is stimulated by thyrotropin-releasing hormone (TRH) from hypothalamus.[3] Thyroid hormones play a crucial role in regulating metabolism, growth of individual and brain development as well as catabolism of biomolecules.[3, 4]

The liver and thyroid hormones engage in multiple levels to maintain homoeostasis. The liver depends on sufficient levels of thyroid hormones to perform its metabolic function, and deficiency in thyroid hormones can lead to systemic effects, including impaired liver function.[5] The liver is essential for regulating thyroid hormone activity, as it activates and deactivates these hormones through the seleno-deiodinase enzyme system. Additionally, the liver supports the bioavailability of thyroid hormones by producing transport proteins such as thyroxine-binding globulin, transthyretin, and albumin. In turn, thyroid hormones influence various liver functions.[6, 7] Several studies have indicated abnormal levels of liver enzymes such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) in patients with hypothyroidism.[8, 9]

These biochemical changes may serve as early indicators of hepatic involvement and can guide better clinical management of hypothyroid patients as in case of hypothyroidism, a derange in liver function tests (LFT) has not been studied in our setting. Therefore, this study aims to evaluate the patterns of LFT in patients diagnosed with hypothyroidism at Kathmandu Medical College Teaching Hospital.

Methods

This analytical cross sectional study was conducted in Kathmandu medical college at medicine department from 1st August to 30th November 2025, after obtaining ethical clearance from institutional review committee (IRC reference No. 25052025/16). In this study, 145 hypothyroid and the same number of euthyroid subjects with their age ranging from 20-60 years were enrolled in the study. They were diagnosed by clinician with laboratory tests, such as TFT (fT3, fT4 and TSH) and LFT (total bilirubin, direct bilirubin, total protein, albumin, ALT, AST, ALP, Gamma Glutamyltransferase- GGT) as well as by physical examination. A convenient consecutive sampling technique was used for data collection. The participants were informed about the objective of the study and oral and written consents were taken from them. A venous blood sample was collected in a plain vial, serum was separated after centrifugation and LFT were analysed using Beckman Coulter Diagnostics automated analyser and TFT were analysed using Siemens analyzer by chemiluminescence immunoassays (CLIA) method.

Grouping of study participants was done as Euthyroidism and Hypothyroidism, the latter was further categorized as subclinical hypothyroidism (SCH) and overt hypothyroidism (OH)

The reference ranges for LFT and TFT were considered as: serum total bilirubin: 0.2-1.0mg/dl, direct bilirubin: 0-0.4mg/dl, indirect bilirubin: 0.2-0.7 mg/dl and is assessed by modified Jendrassik-Groff method, total protein: 6.0-8.0 g/dl is estimated by modified biuret methods, albumin: 3.5-5 g/dl is measured by Bromocresol green (BCG) method , globulin: 2.5-3.5g/dl and is calculated by deducting albumin from total protein. AST: 0-40 IU/L, ALT: Male:0-40 IU/L, Female:0-35IU/L, ALP: 40-125 IU/L, GGT: 5-55 IU/L is estimated by kinetic methods, fT3: 2.02-4.43 pg/ml, fT4: 9.3-17.1 pg/ml, TSH: 0.27-4.20 uIU/ml measured by chemiluminescence immunoassays (CLIA) method.[10-12] Patients were categorized as hypothyroid and euthyroid based on the reference ranges of TFT. Furthermore, overt hypothyroidism was diagnosed as high TSH but low fT3 and fT4 levels. Similarly, sub-clinical hypothyroidism was diagnosed as high TSH with normal fT3 and fT4 levels.[13]

Inclusion criteria: Participants diagnosed as hypothyroidism based on TSH, fT4 and fT3 with their age ranging from 20–60 years.

Exclusion criteria: Participants with any history of liver diseases such as hepatitis, cirrhosis,

fatty liver or cholestasis and any conditions that interfere with LFT, such as individuals with bone and muscle disease, metabolic diseases for instance diabetes mellitus and hypertension, alcoholism and patients with active infection were excluded from the study. Similarly, participants taking hepatotoxic substances like phenylbutazone, aspirin, steroids and amiodarone, lithium and propylthiouracil were not included in the study.

The sample size was determined using two-sample mean difference formula from the study.[9]

$$n = \frac{2X\,(Z\,a/2 + Zb)^2\,X\,\sigma^2}{\Delta^2}$$

Where,

$Z\alpha/2 = 1.96$ at 95% of Confidence Interval (CI)

$Z\beta = 0.80$ (80% power)

σ = pooled standard deviation

Δ = absolute difference in group mean

So, using following information from the study where, ALT mean and standard deviation for control and hypothyroidism are as follows,

$Mean_1 = 25.73$, $S.D_1 = 10.12$,

$Mean_2 = 40.10$, $S.D_2 = 57.97$

$$\sigma = \frac{\sqrt{(Sd1)^2 + (Sd2)^2}}{2}$$

$\sigma = 41.61$

While $\Delta = mean_1 - mean_2 = 14.37$

So putting the values

$$n = \frac{2 \times 7.84 \times (41.61)^2}{14.37^2}$$

$= 27136.68/206.52$

$= 131.4$ in each group

So putting non-response rate of 10%

$131.4 \times 10\% = 13.14$

So the sample size would be $131.4 + 13.14$

$= 144.54$

So, for this study 145 participants will be taken as a hypothyroidism and euthyroidism.

Data were entered in Microsoft Excel and then it was analyzed by Statistical Package for Social Service (SPSS) for windows version 21. Categorical data were expressed in percentage and frequency. Numerical data were expressed as median and interquartile range (IQR). Since the numerical data deviated significantly from normality, as depicted by the Shapiro-Wilk test, non-parametric tests were used for the analysis. Specifically, the Mann-Whitney U test (MWU test), Kruskal-Wallis test (or H test), Post-hoc testing strategy (Bonferroni) and Spearman's correlations were performed where; $p\ value < 0.05$ was considered statistically significant.

Results

Out of 290 participants, 53(36.6%) male and 92(63.4%) female were hypothyroid subjects whereas 64(44.1%) male and 81(55.9%) female were euthyroid group. The median age of hypothyroid subjects was 43 years (35-51 years) and it was 40 years (34.5-48.5 years) among euthyroid group. The participants were categorized into four sub-groups according to their age which is shown in Table 1.

Table 1. Age and gender wise distribution of participants

Variables		Hypothyroidism, n=145 (%)	Euthyroidism, n=145 (%)
Age Groups (Years)	20-30	20 (13.8%)	20 (13.8%)
	31-40	41 (28.3%)	53 (36.6%)
	41-50	47 (32.4%)	48 (33.1%)
	51-60	37 (25.5%)	24 (16.6%)
Gender	Male	53 (36.6%)	64 (44.1%)
	Female	92 (63.4%)	81 (55.9%)

LFT and TFT were assessed in both the groups, each comprising 145 participants. Serum levels of total bilirubin and albumin were significantly higher in hypothyroidism compared to that in euthyroidism. The total protein and globulin were significantly lower in hypothyroidism compared to that in euthyroidism. Serum levels of aminotransferases (ALT and AST) were significantly higher in hypothyroidism when compared to euthyroidism. In contrast, serum ALP and GGT were decreased in hypothyroidism compared to that in euthyroidism which is shown in Table 2.

Table 2. Comparison of liver function and thyroid function tests between hypothyroidism and Euthyroidism

Parameters	Hypothyroidism (Median, IQR), (n=145)	Euthyroidism (Median, IQR), (n=145)	U Statistic	p-value (MWU test)
Total Bilirubin (mg/dl)	0.62(0.51-0.79)	0.60(0.48-0.68)	8758.00	0.014
Direct Bilirubin (mg/dl)	0.10(0.08-0.14)	0.10(0.08-0.15)	10453.50	0.934
Total protein (g/dl)	6.90(6.50-7.27)	7.09(6.67-7.42)	8945.50	0.028
Albumin (g/dl)	4.3(4.12-4.54)	4.01(3.60-4.36)	6381.50	<0.001
Globulin (g/dl)	2.54(2.17-3.03)	3.00(2.70-3.29)	5792.50	<0.001
ALT (IU/L)	23.78(18.00-31.50)	21.70(18.64-24.56)	8802.00	0.017
AST (IU/L)	26.12(20.77-32.00)	24.54(21.29-26.79)	8880.00	0.022
ALP (IU/L)	72.00(59.50-83.61)	76.90(67.27-90.94)	8286.00	0.002
GGT (IU/L)	28.00(21.44-41.80)	34.40(27.52-42.00)	8608.00	0.008
fT3 (pg/ml)	3.17(2.49-3.45)	2.94(2.62-3.15)	9099.50	0.048
fT4 (pg/ml)	11.20(9.35-13.20)	10.55(9.65-11.80)	9683.00	0.245
TSH (uIU/ml)	7.81(5.77-23.00)	2.37(1.60-3.29)	44.00	<0.001

p-values obtained from Mann-Whitney U test. $p< 0.05$ considered statistically significant

A Kruskal–Wallis test showed statistically significant differences in serum total bilirubin, albumin globulin, ALP, GGT, fT3, fT4 and TSH among subclinical hypothyroidism (SCH), overt hypothyroidism (OH) and euthyroidism. Post-hoc pairwise comparisons with Bonferroni correction revealed that serum total bilirubin level was significantly higher in OH when compared with euthyroidism. Serum globulin was significantly higher in euthyroidism when compared with SCH and OH. Serum ALP and GGT were lower in SCH when compared with euthyroidism. Serum albumin, fT3, fT4 and TSH levels differed significantly across all three groups, with lowest levels for albumin and TSH among euthyroidism which is shown in Table 3.

Table 3. Comparison of liver and thyroid parameters between Subclinical, Overt hypothyroidism and euthyroidism

Parameter	SCH Median (IQR), (n=109)	OH Median (IQR), (n=36)	Euthyroidism Median (IQR), (n=145)	H Statistics	p-value (H test)	Post-hoc (bonferroni-corrected
Total Bilirubin (mg/dl)	0.62 (0.51-0.79)	0.68 (0.53-0.85)	0.60 (0.48-0.68)	7.047	0.029	OH vs Euthyroid
Direct bilirubin (mg/dl)	0.11 (0.08-0.15)	0.10 (0.09-0.12)	0.10 (0.08-0.15)	0.293	0.864	-
Total protein (g/dl)	6.90 (6.51-7.35)	7.00 (6.50-7.20)	7.09 (6.67-7.42)	4.834	0.089	-
Albumin (g/dl)	4.29 (4.12-4.49)	4.50 (4.14-4.77)	4.01 (3.60-4.36)	37.814	<0.001	All pair wise comparision
Globulin (g/dl)	2.62 (2.24-3.02)	2.40 (1.82-3.08)	3.00 (2.70-3.29)	44.895	<0.001	OH vs euthyroid, SCH vs euthyroid
ALT (IU/L)	23 (18-31.50)	24 (19.07-34)	21.70 (18.64-24.56)	5.949	0.051	-
AST (IU/L)	26.12 (20.85-32)	27 (20.21-32)	24.54 (21.29-26.79)	5.229	0.073	-
ALP (IU/L)	71 (55.29-80)	76 (67.50-88.75)	76.90 (67.27-90.94)	15.040	0.001	SCH vs euthyroid
GGT (IU/L)	28 (21.27-40.85)	28.27 (21.50-43.48)	34.40 (27.52-42.00)	7.183	0.028	SCH vs euthyroid
fT3 (pg/ml)	3.28 (3.04-3.51)	1.80 (1.58-1.98)	2.94 (2.62-3.15)	106.924	<0.001	All pair wise comparision
fT4 (pg/ml)	12.20 (10.65-13.70)	7.1 (5.42-8.57)	10.55 (9.65-11.80)	121.278	<0.001	All pair wise comparision
TSH (uIU/ml)	6.4 (5.40-8.78)	100 (42.65-100)	2.37 (1.60-3.29)	232.073	<0.001	All pair wise comparision

p-values obtained from bonferroni-correction (adjusted $p<0.016$ is statistically significant)

Spearman's correlation analysis revealed a positive significant correlation of serum albumin as well as ALP with TSH whereas, ALT, AST and Bilirubin showed an insignificant positive correlation with TSH. While, serum total protein and GGT showed a negative insignificant correlation with TSH which is shown in Table 4.

Table 4. Correlation of LFT parameter with thyroid function test

Variables		fT3(pg/ml)	fT4(pg/ml)	TSH(uIU/ml)
Total Bilirubin (mg/dl)	ρ	-0.045	0.078	0.021
	p-value	0.594	0.354	0.804
Total protein (g/dl)	ρ	0.118	0.033	-0.102
	p-value	0.157	0.693	0.224
Albumin (g/dl)	ρ	-0.158	-0.164	0.207
	p-value	0.058	0.049	0.013
ALT (IU/L)	ρ	-0.111	-0.021	0.157
	p-value	0.185	0.798	0.060
AST (IU/L)	ρ	-0.032	0.015	0.113
	p-value	0.703	0.862	0.177
ALP (IU/L)	ρ	-0.181	-0.173	0.208
	p-value	0.030	0.037	0.012
GGT (IU/L)	ρ	-0.016	-0.098	-0.076
	p-value	0.851	0.240	0.363

p-values obtained from Spearman's correlations. $p< 0.05$ considered statistically significant

Discussion

In this study, among 145 hypothyroidism, 92 (63.4%) participants were females showing female predominance and the maximum number of participants were in age group of 41-60 years which could be due to increase age, hormonal fluctuation associated with menopause, genetics and environmental factors and higher predisposition to autoimmune disease especially Hashimoto's thyroiditis which results into less effective immune system in females. These variations changed the secretion of thyroid hormones and hence their serum levels.[14] Thyroid hormones strongly influence the oxygen use and regulate the metabolic activity of virtually all cells, including the hepatocytes.[15] In this study, serum liver enzymes (AST, ALT), protein, albumin and bilirubin level were higher in hypothyroidism while ALP and GGT activity were lower in hypothyroidism when compared to euthyroidism.

Finding of this study is consistent with various other studies which reported increased bilirubin level in hypothyroidism.[14, 16] Since, adequate circulating thyroid hormones plays a crucial role in regulating liver function and bilirubin metabolism.[9] In case of hypothyroidism, activity of bilirubin UDP-glucuronyltransferase is decreased which results in a reduction in bilirubin excretion resulting into hyperbilirubinemia.[16, 17] Present study showed an increased levels of bilirubin in OH than in SCH which is also supported by other studies.[14, 18] This study depicted an insignificant and positive correlation of serum bilirubin with TSH in hypothyroidism which is similar to another study.[19]

Present study found a decrease in serum total protein in hypothyroidism compared to euthyroidism. The decrease level of serum protein in hypothyroidism

could be due to chronic impairment in hepatic function, which causes decreased hepatic protein synthesis due to low thyroid hormone-mediated metabolic activity.[20, 21] Even more, comparing among hypothyroidism, serum total protein was marginally decreased in SCH when compared with OH and was statistically insignificant. It could be due to mild elevation of TSH with normal fT3 and fT4 level for a longer duration.

In this study serum albumin was slightly higher in hypothyroidism compared to that in euthyroidism but remained within normal range which could be due to reduced catabolism resulting prolonging of albumin half-life. The thyroid hormones play an important role in albumin turnover, so reduced hormonal levels in hypothyroidism leads to decrease albumin degradation.[14, 22] The comparison of serum albumin between SCH and euthyroidism; OH and euthyroidism as well as SCH and OH was statistically significant with Post-hoc (bonferroni-corrected, $p<0.016$). The decrease serum albumin observed in SCH compared to OH may be explained by early impairment of hepatic albumin synthesis in the presence of elevated TSH. Since, in OH, albumin degradation is markedly reduced, leading to prolongation of albumin half-life and relative preservation of serum albumin level. Thus, such differences in albumin synthesis, degradation and distribution might lead for such variation in hypothyroidism.

This study showed a positive significant correlation of serum albumin with TSH and negative significant correlation with fT4 and a negative insignificant correlation with fT3 which is in accordance with another study.[23]

This study found an elevated serum aminotransferases activity. Among the hypothyroid patients, 19(13.1%) had a raised ALT activity and 13(9%) had a raised AST activity. An increment in aminotransferase occur due to alteration in metabolism of lipids, hypothyroid myopathy or because of hepatic steatosis.[8] Similar finding were reported in other study.[24] ALT and AST activities were increased in OH when compared to SCH but was statistically insignificant. Various other studies reported statistically significant rise in ALT and AST activities in OH when compared with SCH.[14, 18] Such raised in ALT and AST activities in SCH and OH could be due to myopathy associated with hypothyroidism.[16, 24, 25] Similar to a study,[19] this study found an insignificant positive correlation of serum ALT and AST with TSH. Some other studies depicted positive and significant correlation of serum ALT and AST with TSH.[14, 18] Liver function tests are essential for determining the functional status of the liver and assessing the hepatotoxic effects of external substances. Hepatocellular injury is commonly indicated by elevated serum levels of ALT, AST and ALP. ALT is an aminotransferase which is found throughout the body but is predominantly located in the cytosol of hepatocytes. It is more specific as well as a more sensitive marker of hepatocellular injury than AST. AST is also an aminotransferase which is widely distributed in various tissues of heart, skeletal muscle, liver, etc. In hepatocytes, it is present in the cytosol and mitochondria. Both transaminases are tested as a part of liver panel to assess liver health, with elevated level pointing to issues like hepatitis, fatty liver or alcohol damage.[26, 27]

Thyroid hormones, such as fT3 and fT4 are essential for the growth, development, and normal functioning of all body organs. These regulate the basal metabolic rate of all cells and influence hepatic function too. The liver, in turn, is responsible for the metabolism of thyroid hormones and modulation of their systemic endocrine actions. As a result, thyroid dysfunction can impair liver function, while liver disease can alter thyroid hormone metabolism.[25]

In this study, serum ALP activity was decreased in hypothyroidism than that in euthyroidism which was consistent with other studies.[24, 28] It is mainly due to reduced bone turn over since ALP is largely derived from bone and liver and thyroid hormones are responsible for the bone formation and remodeling. Serum ALP is an enzyme present in most organs of the body and is especially associated with cell surface located in the mucosa of the small intestine, in bones (osteoblasts), liver and placenta. Elevated level of ALP in serum often signals liver issues or bone problem while lower level can point to deficiencies or diseases of liver and bone health.[27] Furthermore, in hypothyroidism osteoblastic activity is reduced leading to decrease bones derived ALP resulting to decrease ALP. Among hypothyroidism, serum ALP activity was increased but statistically insignificant in OH when compared to SCH.

A study also reported similar finding but it was statistically significant.[14] The comparison of serum ALP between SCH and euthyroidism was statistically significant. Similarly, ALP activity showed a positive significant correlation with TSH which was consistent with various other studies.[9, 18] The significant positive correlation could be due to Hypo functioning of thyroid gland which alters membrane lipid composition by increasing the cholesterol-to-phospholipid ratio and reducing membrane fluidity. These changes can impair the functioning of canalicular membrane transporters and enzymes, including Na^+/K^+-ATPase, leading to altered ALP activity.[25]

A decrease in GGT activity was obtained in hypothyroidism in present study which is consistent with the similar study.[28] As thyroid hormones play an important role in the synthesis and regulation hepatic microsomal enzyme, the GGT. Therefore, the reduced level of circulating thyroid hormones leads to reduced hepatic enzyme synthesis and metabolic activities. GGT is the liver peptidase and is a sensitive indicator of the presence of hepatobiliary disease. Similar to ALP, GGT activity is increased in cases of intrahepatic or posthepatic biliary obstruction. An increase in GGT activity is also found in the sera of patients with alcoholic hepatitis and in the majority of sera from people who are heavy drinker.[26, 27]

The comparison of serum GGT between SCH and euthyroidism was statistically significant. But GGT shows negative insignificant correlation with TSH. It could be due hypo functioning of thyroid hormone where high TSH may suppress hepatic GGT synthesis. Additionally, other metabolic and liver factors like fatty liver, oxidative stress or cholestasis condition can influence GGT independently, contributing to an inverse relationship with TSH.

LFT are routinely used to assess hepatic status and provide valuable diagnostic and prognostic information in both clinical practice and occupational medicine. Understanding the association between hypothyroidism and alterations in liver biochemical markers is important, as thyroid dysfunction can influence hepatic enzymatic, degradative, and synthetic functions. Nevertheless, further studies are required to clarify the long-term effects of hypothyroidism on hepatic function.

Limitation of this study was other liver function test like, 5'ncleotidase, Prothrombin Time and Lactate Dehydrogenase (LDH), other diagnostic tests like AntiTPO, lipid profile, c-reactive protein (CRP), creatinine phosphokinase (CPK) were not performed, because of the mostly out-of-pocket payment system. The inclusion of hypothyroid patients irrespective of disease duration and levothyroxine treatment status may influence TSH level and liver enzyme activities, potentially acting as a confounding factors. Due to the cross-sectional study and limited availability of detail treatment history, stratified or adjusted analyses based on treatment status and duration could not be performed. Due to limited resources and time constraint; this study couldn't include other marker to determine their extent of dysfunctions and to confirm our findings. Therefore, further studies are required to be carried out in large sample including various diagnostic tests. Since this study was conducted in tertiary care hospital, the data cannot be extrapolated on the general population.

Conclusion

The findings of this study ascertained that hypothyroidism is a common metabolic disorder showing an increase in serum total bilirubin that was more pronounced in overt hypothyroidism. Hypothyroidism showed a reduction in serum total protein suggesting chronic impairment of hepatic protein synthesis. Although serum ALT and AST levels were elevated in hypothyroid subjects, they showed a positive but insignificant correlation with TSH. A decrease in serum ALP and GGT concluded no evidence of significant hepatocellular or cholestatic liver damage. Overall, the observed correlation pattern indicates that increasing TSH levels are associated with subtle, thyroid-related changes in hepatic biochemical parameters. Since, hypothyroidism showed hepatic metabolic changes, early evaluation of liver function test in thyroid disorder help assess functional status of liver and hepatotoxic effects. Early identification and correction of thyroid hormone deficiency may help prevent subsequent impairment of liver function.

Author Contribution

Concept design: AD, AP; Literature search: AD, JMT, SP; Data collection: AD, SP; Data analysis:

AD; Draft manuscript: AD, AP, JMT; Final manuscript and accountability: All

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Conflict Of Interest

None

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