DOI: https://doi.org/10.54393/pjhs.v6i9.3339

PAKISTAN JOURNAL OF HEALTH SCIENCES
(LAHORE)
https://thejas.com.pk/index.php/pjhs
ISSN (E): 2790-9352, (P): 2790-9344
Volume 6, Issue 09 (September 2025)

Original Article

OPEN ACCESS

Association of Liver Enzymes with Thyroid Hormone Levels in Hyperthyroid Patients: A Cross-Sectional Study from a Tertiary Care Hospital

Faiza Javaid[1], Ahamd Raza Khan[2*], Hamza Khan Sherwani[2], Asma Rasheed[3], Maleeha Sadiq[2] and Isra Asif[2]

[1]Department of Pathology, Al-Aleem Medical College, Gulab Devi Educational Complex, Lahore, Pakistan
[2]Department of Allied Health Sciences, Gulab Devi Educational Complex, Lahore, Pakistan
[3]Department of Pathology, University of Lahore, Lahore, Pakistan

ARTICLE INFO

Keywords:
Thyroid Stimulating Hormone, Triiodothyronine, Liver Function Tests, Alanine Aminotransferase, Aspartate Aminotransferase, Alkaline Phosphatase

How to Cite:
Javaid, F., Khan, A. R., Sherwani, H. K., Rasheed, A., Sadiq, M., & Asif, I. (2025). Association of Liver Enzymes with Thyroid Hormone Levels in Hyperthyroid Patients: A Cross-Sectional Study from a Tertiary Care Hospital: Association of Liver Enzymes with Thyroid Hormone Levels in Hyperthyroid Patients. Pakistan Journal of Health Sciences, 6(9), 59-63, https://doi.org/10.54393/pjhs.v6i9.3339

***Corresponding Author:**
Ahmad Raza Khan
Department of Allied Health Sciences, Gulab Devi Educational Complex, Lahore, Pakistan
kahmadraza14@gmail.com

Received Date: 1st July, 2025
Revised Date: 2nd September, 2025
Acceptance Date: 9th September, 2025
Published Date: 31st September, 2025

ABSTRACT

Hyperthyroidism, characterized by the overproduction of thyroid hormones, may impact liver function. Understanding this relationship is essential for early identification and management of liver dysfunction in hyperthyroid patients. **Objectives:** To determine the association between thyroid hormone levels (TSH, T3, T4) and liver function tests (ALT, AST, ALP, bilirubin, and total protein) in patients with hyperthyroidism. **Methods:** This cross-sectional study was conducted from 28 July 2023 to 28 January 2024 at the Endocrinology OPD of Gulab Devi Hospital, Lahore. A total of 100 hyperthyroid patients were selected using non-probability sampling. Demographic data, thyroid profiles (T3, T4, TSH), and liver function tests (LFTs) were recorded. Data were analyzed by SPSS version 26.0 using descriptive statistics and Chi-square tests to assess associations. **Results:** Out of 100 hyperthyroid patients, 59 were female and 41 were male, with a mean age of 38.5 + 10.2 years. Elevated ALT was observed in 27% of patients, AST in 13%, ALP in 23%, bilirubin in 13%, and total protein in 11%. Chi-square analysis showed significant associations between TSH and ALT (p=0.028) and AST (p=0.017), as well as between T3 and ALP (p=0.031). No significant associations were found between T4 and any of the liver enzymes. **Conclusions:** A significant proportion of hyperthyroid patients showed abnormal LFTs, indicating a relationship between thyroid dysfunction and hepatic involvement. Further large-scale studies are recommended.

INTRODUCTION

Excessive secretion and release of thyroid hormones from the thyroid gland, leading to abnormally elevated blood levels, is called hyperthyroidism [1]. The hypothalamus produces thyroid-releasing hormone, which stimulates the pituitary gland to produce thyroid-stimulating hormone (TSH). This, in turn, stimulates the thyroid gland to produce thyroxine (T4) and triiodothyronine (T3). Increased thyroid hormone secretion inhibits the hypothalamus and pituitary from releasing thyroid-releasing hormone and TSH, respectively [2]. T3 is the biologically active thyroid hormone that binds to thyroid hormone receptors (TR), whereas T4 is a prohormone that must be transformed into T3 before signaling and biological activity can occur [3]. Excess thyroid hormone accelerates the body's metabolic processes, producing clinical manifestations such as tachycardia, weight loss, heat intolerance, muscle weakness, tremors, and sleep disturbances [4]. Beyond these systemic effects, hyperthyroidism also has profound implications for liver physiology and pathology. The liver is a crucial organ responsible for numerous functions related

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to metabolism, digestion, detoxification, storage, and the regulation of vital substances. It plays a dual role in thyroid physiology: it metabolizes thyroid hormones through glucuronidation, sulfation, and iodination [5], and at the same time, hepatic metabolic functions are dependent on adequate thyroid hormone levels. This bidirectional relationship makes the liver particularly vulnerable to thyroid dysfunction. In hyperthyroidism, excessive thyroid hormone increases basal metabolic rate and hepatic oxygen consumption, predisposing the perivenular regions of the liver to relative hypoxia and subsequent injury [6]. Other mechanisms include direct hepatotoxicity from hepatocyte anoxia, oxidative stress and free radical damage, breakdown of hepatic glycogen and proteins, and autoimmune-mediated liver injury. Furthermore, several studies indicate that excess T3 induces hepatocyte apoptosis through mitochondria-dependent pathways and activates death receptor-mediated signaling, thereby exacerbating liver dysfunction [7]. Recent evidence highlights that thyroid dysfunction not only alters liver enzyme patterns but may also contribute to the progression of chronic liver diseases and influence treatment outcomes [8]. However, data from local populations remain scarce, underscoring the need for region-specific studies to better understand the clinical significance of these biochemical alterations. The novelty of this study lies in its focus on a local patient population, where limited data are available regarding the biochemical alterations in hyperthyroidism. By characterizing these changes in liver enzyme patterns, the study provides region-specific evidence that may contribute to improved diagnostic evaluation and clinical management strategies. This study aims to ascertain the level of liver function tests in the serum of patients with hyperthyroidism and to investigate the relationship between liver damage and hyperthyroidism.

METHODS

This cross-sectional study was conducted over six months from 28 July 2023 to 28 January 2024 at the Endocrinology OPD of Gulab Devi Hospital. Ethical clearance was obtained from the Institutional Review Board (Ref. No. GPMI/AHS/IRB 15623), ensuring participant confidentiality and the right to withdraw without consequence. Informed consent was obtained in writing from all participants after explaining the procedure, purpose, risks, and benefits of the study. This study included a sample size of 100 people. 3ml serum of hyperthyroid patients was collected and processed for LFTs in the Pathology lab of Gulab Devi Hospital, Lahore. The sample size was calculated by using Cochran's formula[9].

$$n = \frac{Z^2 \frac{\alpha}{2} pq}{\rho^2}$$

n =sample size, P = prevalence, z = confidence, q = 1-p, ρ = margin of error. For this study, P was set at 55%, based on previous literature reporting that approximately 55% of hyperthyroid patients exhibit liver enzyme abnormalities. The margin of error (ρ) was set at 6%, with a 95% confidence interval. Using these values, the calculated sample size was approximately 264 participants. Due to the six-month duration of the study, during which informed consent, ethical approval, and patient data were collected, the sample size was limited to 100. This was influenced by the restricted availability of patients meeting the inclusion and exclusion criteria, as well as constraints in resources and time. Patients aged between 18-60 years of both genders, diagnosed with hyperthyroidism, were included. Patients below 18 or above 60 years of age, those with a history of alcoholism, chronic liver disease, and those with known hepatitis were excluded. The demographic data, such as age, gender, and marital status, were collected through a questionnaire and verified against hospital records. Blood samples (3 ml) were drawn from each patient to evaluate both liver function tests (LFTs) and thyroid hormone levels (TSH, T3, and T4). The analyses were performed using standard biochemical assays on the Micro Lab 400 instrument, which operates on the principle of spectrophotometry, at the Chemical Pathology Laboratory of Gulab Devi Hospital, Lahore. The reference (cut-off) values used to classify enzyme and hormone levels as high, normal, or low were as follows: ALT: 0–42 U/L, AST: 0–45 U/L, ALP: 44–147 U/L, total bilirubin: 0.1–1.1 mg/dL, total protein: 6.5–8.3 g/dL, TSH: 0.4–4.0 mIU/L, T3: 80–200 ng/dL, and T4: 5.0–12.0 µg/dL (10). These cut-off values were applied to categorize patient results and assess associations with thyroid hormone levels using Chi-square tests with significance at a p-value of <0.05 and descriptive statistics such as mean, median, mode, and standard deviation. The Chi-square test was chosen because it is appropriate for assessing the association between categorical variables, such as thyroid hormone levels and liver function test categories. Qualitative data was presented in the form of charts, and quantitative data was presented in the form of tables and graphs. All collected data were statistically analysed by using SPSS version 26.0.

RESULTS

Out of 100 cases, 41% (n=41) were male and 59% (n=59) were female. A total of 21% (n=21) were aged 34–40 years. The mean age was 38.5 ± 10.2 years (range: 18–60 years). Most patients were married (87%, n = 87). The mean height was 5.51 ± 0.39 ft, and the mean weight was 66.39 ± 14.67 kg. The

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mean systolic blood pressure was 126.05 ± 14.9 mmHg, and the mean diastolic blood pressure was 90.14 ± 10.79 mmHg. A family history of hyperthyroidism was present in 25% (n=25), while 39% had a history of hypertension. The biochemical parameters were as follows: mean TSH 13.35 ± 6.02 mIU/L, mean T3 152.92 ± 40.26 ng/dL, mean T4 23.6 ± 10.60 µg/dL, mean AST 43.37 ± 35.68 U/L, mean ALP 127.70 ± 40.37 U/L, mean ALT 59.37 ± 45.00 U/L, mean total bilirubin 0.70 ± 0.30 mg/dL, and mean total protein 7.9 ± 0.8 g/dL (Table 1).

Table 1: Demographic, Clinical, and Biochemical Characteristics of Patients

Characteristics	Mean ± SD	Median	Mode
Age	38.5 ± 10.2	40	40
Height	5.51 ± 0.39	5.50	5.00
Weight	66.39 ± 14.67	65	60
Systolic BP	126.05 ± 14.9	120	120
Diastolic BP	90.14 ± 10.79	90	90
TSH	13.35 ± 6.02	11.5	10
T3	152.92 ± 40.26	111.6	94
T4	23.6 ± 10.60	10.7	7
AST	43.37 ± 35.68	25	18
ALP	127.70 ± 40.37	92	90
ALT	59.37 ± 45.00	28	27
Bilirubin	0.70 ± 0.3	0.60	0.50
Total Protein	7.9 ± 0.8	7.20	7.50

By applying the chi-square (χ^2) test between T3 and LFTs, a significant association was found between T3 and ALP (p=0.031). No significant associations were observed with ALT, AST, bilirubin, or total protein (p>0.05) (Table 2).

Table 2: Cross-tabulation of Tri-iodothyronine (T3) with LFTs

		T3			χ^2	p-Value	Cramer's V
Parameters	Category	>180 (High)	<180 (Normal)	<60 (Low)			
AST	>45 (High)	3	8	2	2.334	0.311*	0.153
	<45 (Normal)	23	60	4			
ALP	>147 (High)	13	12	2	10.363	0.031*	0.231
	<147 (Normal)	13	54	4			
	<44 (Low)	0	2	0			
ALT	>42 (High)	9	15	3	3.218	0.200*	0.179
	<42 (Normal)	17	53	3			
Bilirubin	>1.1 (High)	4	7	2	3.182	0.528	0.165
	<1.1 (Normal)	22	60	4			
Total Protein	>8.3 (High)	2	9	0	1.376	0.502	0.117
	<8.3 (Normal)	24	59	6			

When the chi-square test was applied between TSH and LFTs, significant associations were observed with ALT (p=0.028) and AST (p=0.017). No significant associations were found with ALP, bilirubin, or total protein (p>0.05) (Table 3).

Table 3: Cross-Tabulation of Thyroid-Stimulating Hormone (TSH) With LFTs

Parameters		TSH			χ^2	p-Value	Cramer's V
		High	Normal	Low			
ALT	High	4	5	18	7.160	0.028*	0.268
	Normal	2	7	64			
AST	High	3	2	8	8.168	0.017*	0.286
	Normal	3	10	74			
ALP	High	2	3	22	0.591	0.964	0.054
	Normal	4	9	58			
	Low	0	0	2			
Bilirubin	High	2	2	9	2.812	0.590	0.119
	Normal	4	10	72			
	Low	0	0	1			
Total Protein	High	1	2	8	7.20	0.698	0.085
	Low	5	10	74			

No significant associations were observed between T4 and any LFT parameters (p>0.05). For parameters with small cell counts, ALP and bilirubin low categories (n=1–2), Fisher's exact test was applied instead of the chi-square test to ensure valid statistical inference.

DISCUSSIONS

Thyroxine and triiodothyronine regulate hepatocyte metabolic rate and are required for appropriate organ growth, development, and function. In contrast, the liver is responsible for thyroid hormone metabolism and regulates its systemic effects [11]. The pituitary, heart, liver, and brain are all impacted by thyroid hormones T4, T3, and TSH [12]. In current study, we took 100 diagnosed hyperthyroid patients to check the association of elevated liver enzymes with hyperthyroidism. This study found significant associations between elevated TSH/T3 and liver enzymes ALT, AST, and ALP. In particular, the significant associations between TSH and ALT/AST may reflect the pathophysiological effect of thyroid dysfunction on hepatocellular metabolism. Excess thyroid hormones can increase hepatic oxygen demand, leading to oxidative stress and hepatocyte injury, which is commonly reflected in elevated transaminases [13]. While Hsieh *et al.* and Zhang

DOI: https://doi.org/10.54393/pjhs.v6i9.3339

et al. also observed a significant association between thyroid hormones and liver enzymes, their studies involved different patient populations and methodological approaches. Hsieh *et al.* focused on long-term thyroid dysfunction, whereas our study specifically examined newly diagnosed hyperthyroid patients. Similarly, Zhang *et al.* emphasized broader biochemical correlations, while we concentrated on liver enzyme elevation as a direct marker of hepatic involvement [14, 15]. At a biochemical level, hyperthyroidism may accelerate basal metabolic rate, contributing to increased oxygen consumption and oxidative stress in hepatocytes. This oxidative burden, together with altered lipid and carbohydrate metabolism, may play a role in liver cell stress and enzyme release [16]. Moreover, thyroid hormones influence bile acid synthesis and clearance, and their imbalance might impair bile flow, which could contribute to hepatocellular injury [17]. This link illustrates that thyroid diseases, including hypothyroidism and hyperthyroidism, are associated with alterations in liver function, potentially through raised metabolic rate and changes in liver enzyme activity. Such changes may be reflected in increased liver enzyme levels, suggesting possible liver involvement [18]. Interestingly, T3 was significantly associated with ALP, while TSH was not. This may be explained by the fact that T3 is the biologically active hormone directly influencing bone and hepatic enzyme regulation, whereas TSH acts indirectly through stimulation of the thyroid gland. Therefore, ALP elevations in hyperthyroidism may be more directly linked to circulating T3 levels than to TSH [19]. No correlation was observed between T4 and liver enzymes in our study. This may be because T4 is a prohormone requiring conversion to T3 to exert biological activity [20]. Furthermore, the relatively small sample size and categorization thresholds used could have reduced the statistical power to detect subtle associations with T4. Contrary to some studies, which showed no significant differences observed in ALT among patients with overt hyperthyroidism. No significant correlation was found between liver enzymes and thyroid profile in any study group [21]. Current research evaluated all possible relationships between abnormal metabolisms of liver metabolism and hyperthyroidism. This study assessed abnormal liver enzyme activity in relation to thyroid dysfunction in 100 people. Associations between thyroid profile and ALT/AST were significant, while no significant associations were found with Bilirubin, ALP, or Total Protein, which may be more indicative of chronic changes. This study used a non-probability sampling method, which may have introduced some bias and limited the generalizability of the results to the wider population. However, this approach was chosen due to constraints of time and resources, and strict adherence to methodological protocols and carefully applied inclusion and exclusion criteria were implemented, ensuring that the selected participants were representative of the study objectives.

CONCLUSIONS

In this study, significant associations were observed between elevated TSH and ALT/AST, and between T3 and ALP. These findings suggest that hyperthyroidism may be linked to selective liver enzyme abnormalities. Regular monitoring of liver function, particularly ALT, AST, and ALP, could help in early identification and management of potential hepatic involvement in hyperthyroid patients.

Authors Contribution

Conceptualization: FJ, ARK
Methodology: FJ, ARK, HKS, MS, IA
Formal analysis: FJ, ARK, AR, MS
Writing review and editing: FJ, ARK, HKS, AR, IA

All authors have read and agreed to the published version of the manuscript

Conflicts of Interest

All the authors declare no conflict of interest.

Source of Funding

The author received no financial support for the research, authorship and/or publication of this article.

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