Original Article

An Interrelationship of Anti-Thyroid Peroxidase Antibody in Clinical and Subclinical Hypothyroidism

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ABSTRACT

Background: Immune-mediated changes in the thyroid gland cause changes in thyroid hormone levels. These changes lead to autoimmune thyroid disease. Autoimmunity is the main cause of hypothyroidism. Hypothyroidism requires lifelong treatment to avoid health problems related to thyroid hormone deficiency. **Objective:** To evaluate the correlation between Anti-Thyroid Peroxidase Antibody (Anti TPO Ab) and Hypothyroidism. **Materials & Methods:** This cross-sectional study was conducted in the Department of Medicine at Zainul Haque. Sikder Women's Medical College & Hospital from September 2024 to August 2025. Subjects were selected through non-probability, purposive sampling. Based on thyroid profile, the subjects were divided into three groups: euthyroids (n=44), subclinical hypothyroids (n=47), and clinical hypothyroids (n=42). Blood from these subjects was analyzed for Anti-TPO Ab. Data were analyzed using SPSS version 18. **Results:** The study population had a mean age of 38.90 ± 10.69 years for Clinical hypothyroidism, 37.23 ± 12.96 years for subclinical hypothyroidism, and 34.95 ± 14.26 years for euthyroidism. No significant age difference was found across those groups. Serum Thyroid-Stimulating hormone (TSH) was significantly higher ($p < 0.05$) in patients with subclinical hypothyroidism (7.10 µIU/ml) and clinical hypothyroidism (17.15 µIU/ml) than in euthyroid patients (2.21 µIU/ml). FT4 levels were 0.49, 1.10, and 1.20 ng/dL in thyroid, subclinical hypothyroidism, and Euthyroidism, respectively. Anti-Thyroid Peroxidase (Anti-TPO Ab) was 219.5, 37, and 23 IU/mL in Clinical, Subclinical, and Euthyroid subjects, respectively, which was clinically significant. **Conclusion:** The present study concludes that Anti-TPO comes out to be positive in cases of hypothyroid patients and negative in cases of Euthyroid subjects. Autoimmunity is the main cause of hypothyroidism nowadays. In this study, among the Euthyroid group, only three subjects were found to be Anti TPO Ab positive, which indicates they carry a high risk of developing hypothyroidism in the future.

Keywords: Clinical hypothyroidism, Subclinical hypothyroidism, TSH, Anti-TPO Ab.

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Introduction

Thyroid diseases are one of the commonest endocrine disorders in the world. Various studies' results on thyroid disease showed that more than 42 million people suffer from thyroid disorders in South Asia.[1] Clinical Hypothyroidism is defined as a high serum TSH concentration with a decrease in free Thyroxine (FT4) and free triiodothyronine (FT3) concentrations associated with clinical signs and symptoms. Subclinical hypothyroidism (ScHt) is defined as high Serum TSH concentration with normal serum free triiodothyronine (FT3) and free Thyroxin (FT4) concentrations, allied with few or negligible signs and symptoms of hypothyroidism.[2] The prevalence is 9.4% for subclinical hypothyroidism.[3] Clinical presentation may differ from mild and asymptomatic to severe and overt disease; it may depend on the patient's age, gender, physical condition, and the pace at which hypothyroidism develops. Cold intolerance, puffiness, coarse skin, weight gain, decreased physical and mental activity, and periorbital puffiness are the usual signs and symptoms of clinical hypothyroidism, but are seldom seen in subclinical hypothyroidism. Hypothyroidism may be caused by a defect in the thyroid (Primary), pituitary (Secondary), or hypothalamus (Tertiary). Generally, it is primary hypothyroidism caused by iodine deficiency, autoimmunity, and other reasons. Autoimmune thyroid diseases are associated with a number of autoantibodies, which are categorized as either primary or secondary.[4,5] Primary antibodies are a directly useful diagnostic tool for the presence of autoimmune thyroid disease.[6] One of the major secondary antibodies associated with autoimmune thyroid disease is Anti-thyroid Peroxidase Antibodies. The most common autoimmune thyroid disease is Hashimoto thyroiditis, which is characterized by gradual thyroid failure with or without goiter development.[7] Nearly all Hashimoto thyroiditis patients have high serum concentrations of antibodies against one or more thyroid antigens that are produced by lymphocytic infiltrate in the thyroid gland or, to a small extent, by regional lymph nodes or bone marrow. Among general people, the relationship between anti-thyroid antibodies and thyroid profile (TSH, T4, and T3) is not established. In several years, the presence of Anti-TPO antibodies may lead to the progression of clinical thyroid disease.[8] Determining the anti-thyroid antibodies and thyroid profile relation could help identify such a group of patients who have an unstable thyroid profile. Therefore, to rule out underlying autoimmune disease, screening for Anti-TPO antibodies is required. [9] Diagnosis of autoimmune thyroid disorders is important for treatment modifications for autoimmunity, as the presence of autoimmunity almost always indicates a high risk of developing a thyroid disorder in the future. The initiation of autoimmunity can be due to genetic, environmental, stress, infection, or iodine deficiency.[10–12]

Materials and Methods

This cross-sectional study was conducted in the Department of Medicine, Zainul Haque Sikder Women's Medical College Hospital, Dhaka, Bangladesh, between September, 2024 and August, 2025. According to the selection criteria, 89 adult patients (age ≥ 15 years) with hypothyroidism were enrolled in the study. These patients were carefully selected using a purposive sampling technique, a non-random method for selecting participants with specific characteristics. By excluding patients who had experienced an acute myocardial infarction (MI) or acute renal failure within the last three months, pregnancy, hypertension, or acute illness, it was ensured that the study's focus remained on the chronic aspects of Thyroid disease. This exclusion criterion was set to avoid confounding factors that acute conditions could introduce, allowing for a clearer understanding of Hypothyroid's chronic progression and association with a select biochemical profile. After selecting the patients, the aims, objectives, and procedures of the study were explained to the patients in understandable language. Risks and benefits were also made clear to the respondents. The respondents were encouraged to participate voluntarily, and they were allowed to withdraw from the study. Then, written informed consent was obtained from each respondent. Data were collected through face-to-face interviews using a semi-structured questionnaire and data collection tools. The study population underwent detailed history taking, physical examination, and relevant investigations. Serum thyroid-stimulating hormone (TSH) and anti-TPO Ab were assessed for each respondent. Serum TSH, FT4, and Anti-TPO Ab were measured using the CLIA (Chemiluminescent immunoassay) method. Sample size was determined using the equation z^2pq/d^2.

Statistical analysis was performed using Windows-based software, Statistical Package for the Social Sciences 25 (SPSS-25) (Chicago, IL, USA). After collection, all the data were checked and cleaned. Quantitative data were expressed as percentages, means, and standard deviations, and qualitative data were expressed as frequency distributions and percentages. To determine statistical significance, one-way ANOVA, the Kruskal-Wallis test, and the Spearman correlation coefficient test were used

as appropriate. A p-value of less than 0.05 was considered statistically significant.

Ethical approval was received from the ethical review board of Zainul Haque Sikder Women's Medical College journal.

Results

133 were selected for the study and grouped based on thyroid profile and TSH. Among these, 44 subjects were euthyroid, whereas 47 and 42 subjects were in the categories of subclinical and clinical hypothyroid, respectively.

The anti-TPO parameters were compared among euthyroids, subclinical hypothyroids, and clinical hypothyroids. It can be observed from these tables.

Table 1: Demographic profile of the study subjects (N=133)

	Clinical Hypothyroidism	Subclinical Hypothyroidism	Euthyroid (Control)	p-value
n	42	47	44	
Age (years)				
≤20	1 (2.4)	4 (8.5)	8 (18.2)	0.153
21 - 30	10 (23.8)	11 (23.4)	12 (27.3)	
31 - 40	18 (42.9)	14 (29.8)	11 (25.0)	
41 - 50	5 (11.9)	11 (23.4)	4 (9.1)	
>50	8 (19.0)	7 (14.9)	9 (20.5)	
Mean ± SD	38.90 ± 10.69	37.23 ± 12.96	34.95 ± 14.26	0.356
Min-max				
Gender				
Male	3 (7.1)	9 (19.1)	9 (20.5)	0.175
Female	39 (92.9)	38 (80.9)	35 (79.5)	

The ANOVA test was conducted for numerical data, while the Chi-Square test was applied to categorical data. Frequency and percentage were used to present categorical data, while numerical data were expressed as mean accompanied by standard deviation.

Table 2: Thyroid hormone profile of the study subjects (N=133)

	Clinical Hypothyroidism	Subclinical Hypothyroidism	Euthyroid (Control)	p-value
TSH (mIU/mL	17.15 (54.4)	7.10 (2.37)	2.21 (1.28)	<0.001
FT4 (ng/dL)	0.49 (0.38)	1.10 (0.30)	1.20 (0.30)	<0.001
Anti TPO AB (IU/mL)	219.5 (549.0)	37.0 (217.0)	23 (14.95)	<0.001

The Kruskal-Wallis test was conducted, followed by the Holm-Bonferroni adjustment. Data were reported as median accompanied by interquartile range in parentheses.

	TSH	FT4	Anti TPO AB
A vs B	<0.001	<0.001	0.013
A vs C	<0.001	<0.001	<0.001
B vs C	<0.001	0.035	0.002

Table 3: Correlation of TSH with Anti TPO AB and lipid profiles among patients with clinical hypothyroidism (N=133)

		r	p-value
TSH	Anti TPO AB	0.424	0.001

Spearman co-relation coefficient test was done

Discussion

In this cross-sectional study, the age of the study subjects ranged from 15 to 70 years. Anti-TPO Ab of 44 euthyroid subjects was compared and evaluated with 47 subclinical and 42 clinical hypothyroid subjects. Similar results were shown by Jeena et al (2013), who found that the most common thyroid disorder was subclinical hypothyroidism in the study population, followed by overt hypothyroidism.[13] In our study, the reference value of Serum TSH is <4.12 μIU/ml and Anti TPO Ab is <35IU/ml was used according to AACE(2013). Out of 89 subjects, clinical hypothyroid subjects had relatively high TSH levels (TSH > 17.15 μIU/ml). SubClinical hypothyroid subjects had high TSH levels, i.e. >7.10μIU/ml.

A study by Khadka Shrestha Srijana et al. (2017) also showed that subclinical hypothyroidism (56.3%) was more common than overt hyperthyroidism (18.0%), overt hypothyroidism (16.9%), and subclinical hyperthyroidism (8.8%).[14] In the present study, 89 patients out of 133 patients were AntiTPO positive, that is, 66.9%. Similar results were reported by Jeena et al. (2013), who found that 60% of total hypothyroid cases are Anti-TPO-positive. From 42 cases of clinical hypothyroidism, 66.6% of the cases were Anti-TPO positive. 47 cases of subclinical hypothyroidism show 51% Anti - TPO positivity. Similarly, Mohanty et al. showed that 45 of 61 subjects (73.78%) had elevated anti-TPO in subclinical hypothyroid patients.[15] Similarly, Jay Shankar CA et al. (2015) found that 33 of 50 subjects (66%) with either clinical or subclinical hypothyroidism were anti-TPO positive, and 17 were negative. 80% were positive for anti-TPO among the clinical hypothyroidism, whereas patients with ScHt showed (50%) anti-TPO positivity. [16] In this study, an elevated TPO antibody titer was found in the majority of hypothyroid patients. In cases with negative Anti-TPO antibody results, the reason could be that a few still show cytological evidence of autoimmune destruction. The ongoing autoimmune process in the thyroid gland leads to elevated antibody titers. Also, 3 of our euthyroid subjects had positive antibody titers. This may be due to well-compensated thyroid function at present, with a future risk of dysfunction. But, this high prevalence (25%) of positive antibody titer in euthyroid subjects reflects a referral bias, as this sampling was done on subjects attending the hospital. In this study, the mean TSH and Anti-TPO levels were significantly elevated in patients compared with normal controls ($p<0.001$). It is also observed that the FT4 mean is higher in cases than in controls. The past few studies have shown a contradiction regarding FT4. These findings confirm that our study group has higher TSH and Anti-TPO levels. Previous studies by Hollowell J G et al. (1994) showed that anti-TPO antibodies are common in euthyroid subjects.[17-19] Our study shows that the TSH mean value is higher in clinical hypothyroidism than in subclinical hypothyroidism, and Anti-TPO is higher in clinical hypothyroid patients than in subclinical hypothyroid patients. Similar results were shown by Ghoriasian et al and Swain et al. Present studies show that the Anti-TPO-TSH correlation is r = 0.424 in hypothyroid patients. In the past, it showed a positive relation between thyroid function test and anti-TPO antibody levels. Vaseghani et al. found in their research that anti-TPO antibody titers correspond to TSH titers.[20] An important association between TSH or T4 concentration and increased anti-TPO antibody was shown by Ghoraishian et al. A recent study by Thomas Cyriae et al (2015) showed the same results.[21]Studies done in Greece showed that Anti-TPO positive in subclinical hypothyroidism[21], while others showed no significant difference [22.] Limitations of the present study are referral bias and the small sample size. It is a cross-sectional analysis of results. A larger sample with clinical and FNAC correlation is required to validate this test in our population.

Conclusions

The present study concludes that Anti-TPO Ab comes out to be positive>35 IU/ml) In cases of hypothyroid patients, which accounts for about 66.9%, and negative in the case of Euthyroid subjects. Autoimmunity is the main cause of hypothyroidism nowadays. In this study, among the Euthyroid group, only three subjects were found to be Anti TPO Ab positive, which indicates they carry a high risk of developing hypothyroidism in the future.

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