Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Thyroid autoimmunity in euthyroid adults with goiter: frequency of anti-TPO, anti-TG and TRAb positivity in a comparative cross-sectional study from Bangladesh

Satya Prasad Koirala
Bangladesh University
Shahjada Selim
Bangladesh University
Ashraful Hossain
Bangladesh University
Koushik Ashraf
Bangladesh University
Forid Uddin
Bangladesh University
Md. Masud Rana
masud.raj82@gmail.com

Bangladesh University

Research Article

Keywords: Euthyroid goiter, Thyroid autoimmunity, Anti-TPO antibody, Anti-TG antibody, TRAb, Bangladesh

Posted Date: November 20th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11117872/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Abstract

Background

Iodine deficiency has historically been the main cause of goiter in Bangladesh, but autoimmune mechanisms may contribute to thyroid enlargement after salt iodization, even when thyroid function is normal. Data on thyroid autoantibodies in euthyroid Bangladeshi adults with goiter are limited. We compared the frequency of anti-thyroid peroxidase (anti-TPO), anti-thyroglobulin (anti-TG) and thyrotropin receptor (TRAb) antibody positivity in euthyroid adults with and without goiter.

Methods

In this comparative cross-sectional study at the Department of Endocrinology, Bangladesh Medical University, Dhaka (September 2023–March 2026), we enrolled 100 euthyroid adults with goiter and 100 euthyroid adults without goiter. Euthyroidism was confirmed by serum TSH and free thyroxine (FT4) and goiter by ultrasonography. Anti-TPO, anti-TG and TRAb were measured by chemiluminescence immunoassay. Groups were compared using chi-square or Fisher's exact tests and the Mann–Whitney U test; unadjusted odds ratios (ORs) with 95% confidence intervals (CIs) are reported.

Results

Participants with goiter were older (34.3 ± 9.2 vs 31.0 ± 4.9 years) and more often female (80% vs 59%). Anti-TPO positivity was more frequent in the goiter group (36% vs 14%; OR 3.46, 95% CI 1.72–6.94; $p < 0.001$), and anti-TPO titres were higher (median 25.3 vs 8.8 IU/mL). Anti-TG positivity was similar (15% vs 16%; OR 0.93, 95% CI 0.43–1.99; $p = 0.845$). TRAb positivity occurred only in the goiter group (5% vs 0%; $p = 0.059$). At least one antibody was present in 43% of participants with goiter and 20% of those without (OR 3.02, 95% CI 1.61–5.67), and the distribution of antibody patterns differed between groups ($p < 0.001$); however, 57% of participants with goiter were antibody-negative.

Conclusions

In this single-centre sample, anti-TPO positivity, but not anti-TG or TRAb positivity, was more frequent in euthyroid adults with goiter than in those without goiter. The groups differed in age, sex and family history and the estimates were not adjusted, so the findings indicate an association only. Larger, adjusted studies that include iodine status and longitudinal follow-up are needed.

Trial registration:

Not applicable.

Background

Bangladesh has historically carried a heavy burden of iodine deficiency disorders. A national survey in 1993 reported a total goiter rate of 47.1% [1], with goiter graded using WHO/UNICEF/ICCIDD criteria [2]. After the introduction of universal salt iodization, the 2004–05 national survey found total goiter rates of 6.2% in children aged 6–12 years and 11.7% in women aged 15–44 years [3]. Thyroid disorders nevertheless remain common worldwide [4–9] and in Bangladesh [10], and the pattern of disease has shifted from classical iodine-deficiency goiter toward nodular and autoimmune thyroid disease [11].

Goiter is a structural finding rather than a disease entity. It is defined as thyroid enlargement beyond the normal volume for age and sex and can occur in euthyroid, hypothyroid or hyperthyroid states. Euthyroid goiter arises through compensatory glandular enlargement, which may be triggered by iodine deficiency, iodine excess or autoimmune injury to the gland [12]. In iodine-replete settings, autoimmune thyroiditis, characterised by lymphocytic infiltration and circulating thyroid autoantibodies [13, 14], is increasingly recognised as a cause of goiter even when thyroid function is biochemically normal [11].

Regional data suggest a transition from iodine-deficiency goiter toward autoimmune thyroid disease after salt iodization, with rising autoantibody prevalence reported in India [15] and Sri Lanka [16] as iodine status improved. One proposed explanation is that iodine repletion may promote thyroid autoimmunity in genetically susceptible individuals [17, 18]. Thyroid autoantibodies, especially anti-TPO, can also become detectable years before serum TSH or free thyroid hormone concentrations change, creating a clinically silent phase of autoimmune activity [19].

Existing Bangladeshi studies have examined antithyroid antibodies in mixed groups of thyroid patients [20] or in nodular goiter [21]. Data quantifying anti-TPO, anti-TG and TRAb positivity in adults who are biochemically euthyroid but goitrous, relative to euthyroid adults without goiter, remain scarce. We therefore compared the frequency of anti-TPO, anti-TG and TRAb positivity, individually and in combination, between euthyroid adults with goiter and euthyroid adults without goiter attending Bangladesh Medical University.

Methods

Study design and setting

This comparative cross-sectional study was conducted in the Department of Endocrinology, Bangladesh Medical University (BMU), Dhaka, from September 2023 to March 2026. The study is reported in accordance with the STROBE statement for cross-sectional studies [22].

Participants

Adults aged 18 years and above with clinically suspected goiter, and euthyroid adults without goiter, attending the department were enrolled consecutively after written informed consent. Goiter was defined as clinical grade and ultrasound criterion to be stated, e.g., WHO palpation grade and sex-specific thyroid volume cut-offs. Participants in the comparison group were [source of the comparison group and method used to confirm absence of goiter to be stated. All participants had to be biochemically euthyroid (see Laboratory measurements).

The following were excluded: goiter accompanied by thyroid dysfunction or a history of treatment for thyroid dysfunction; diagnosed thyroid malignancy; prior antithyroid drug or radioiodine therapy; acute or chronic illness; current use of medications known to alter thyroid status (e.g., steroids, amiodarone, lithium); pregnancy or lactation; and refusal to give consent.

Sample size

The sample size was calculated for a comparison of two independent proportions, assuming antibody positivity of 19.4% and 6.4% as reported in Bangladeshi thyroid patients and healthy controls, respectively [20], with 95% confidence and 80% power. This gave a minimum of 101 participants per group. Allowing for 5% attrition, 107 participants per group were targeted, and 100 per group were included in the final analysis after exclusions.

Data collection

Demographic data (age, sex, residence, family history of thyroid disease), clinical variables (height, weight, body mass index [BMI], pulse rate, blood pressure, duration and WHO grade of goiter) and laboratory variables (TSH, FT4, anti-TPO, anti-TG and TRAb) were recorded on a structured case record form.

Laboratory measurements

Serum TSH and FT4 were measured on a Maglumi 2000 Plus analyser (Snibe Diagnostics, Shenzhen, China) by chemiluminescence immunoassay. Participants were classified as euthyroid if TSH was 0.3–4.5 μIU/mL and FT4 was 0.9–1.75 ng/dL. Anti-TPO, anti-TG and TRAb were measured on the same platform. Positivity was defined as > 30 IU/mL for anti-TPO, > 95 IU/mL for anti-TG and > 1.5 IU/L for TRAb.

Thyroid ultrasonography

Thyroid ultrasonography was performed at the National Institute of Nuclear Medicine and Allied Sciences (NINMAS), BMU, by a single experienced sonologist to confirm goiter and classify its structural subtype [23].

Statistical analysis

Data were analysed using IBM SPSS Statistics version 29. Categorical variables are presented as frequency and percentage and were compared using the chi-square test, or Fisher's exact test where expected cell counts were below five; for the antibody pattern table, in which many expected counts were below five, an exact (Monte Carlo) test was used. Continuous variables are presented as mean ± SD if normally distributed method of assessing normality or median (25th–75th percentile) if skewed, and were compared using the independent-samples t-test or the Mann–Whitney U test, respectively. Unadjusted odds ratios with 95% CIs (Woolf method) were calculated for antibody positivity; an odds ratio was not estimated where a cell count was zero. A two-sided p-value < 0.05 was considered statistically significant. No adjustment for multiple comparisons was made across the three antibodies, and no multivariable analysis was performed

Results

Study participants

Of 250 individuals assessed, 200 were included: 100 euthyroid adults with goiter and 100 euthyroid adults without goiter. The remaining 50 were not retained, most commonly because they did not meet the biochemical euthyroid criteria or met another exclusion criterion; a smaller number declined consent.

Baseline characteristics

Participants with goiter were older than those without goiter (mean 34.3 ± 9.2 vs 31.0 ± 4.9 years, p < 0.001) and more often female (80.0% vs 59.0%, p = 0.001; Table 1). A family history of thyroid disease (25.0% vs 0.0%, p < 0.001) and diabetes mellitus (8.0% vs 0.0%, p = 0.007) were reported only in the goiter group. BMI category differed between groups (p < 0.001): normal weight was more common in the goiter group, whereas overweight and obesity predominated in the group without goiter. Underweight and class II obesity occurred only in participants with goiter, in small numbers.

Table 1
Baseline characteristics of euthyroid adults with and without goiter

Characteristic	Goiter (n = 100)	No goiter (n = 100)	p value
Age, years, mean ± SD	34.32 ± 9.22	31.03 ± 4.85	< 0.001
Female sex, n (%)	80 (80.0)	59 (59.0)	0.001
Family history of thyroid disease, n (%)	25 (25.0)	0 (0.0)	< 0.001
Diabetes mellitus, n (%)	8 (8.0)	0 (0.0)	0.007

Age was compared using the independent-samples t-test; categorical variables using the chi-square or Fisher's exact test. SD, standard deviation; BMI, body mass index; IQR, interquartile range.

Thyroid function and autoantibody titers

Serum TSH did not differ significantly between groups ($p < 0.077$). FT4 was modestly but significantly lower in the goiter group ($p < 0.002$), although values in both groups remained within the reference range. Anti-TPO titres were markedly higher in the goiter group (median 25.3 vs 8.8 IU/mL, $p < 0.001$), whereas anti-TG ($p < 0.149$) and TRAb titres ($p < 0.334$) did not differ significantly (Table 2).

Table 2
Thyroid function tests and autoantibody titres in euthyroid adults with and without goiter

Variable	Goiter (n = 100) median (IQR)	No goiter (n = 100) median (IQR)	p value
TSH, µIU/mL	2.75 (1.59–3.16)	2.28 (1.48–3.10)	0.077
FT4, ng/dL	1.09 (0.98–1.29)	1.31 (0.99–1.44)	0.002
Anti-TPO Ab, IU/mL	25.32 (19.63–62.25)	8.75 (0.63–25.85)	0.001
Anti-TG Ab, IU/mL	25.00 (20.00–41.65)	23.55 (3.27–46.62)	0.149
TRAb, IU/L	0.58 (0.28–1.02)	0.56 (0.36–0.76)	0.334

Mann–Whitney U test. Euthyroid range: TSH 0.3–4.5 µIU/mL, FT4 0.9–1.75 ng/dL. Positivity cut-offs: anti-TPO > 30 IU/mL, anti-TG > 95 IU/mL, TRAb > 1.5 IU/L. IQR, interquartile range.

Frequency of antibody positivity

Anti-TPO positivity was significantly more frequent in the goiter group than in the group without goiter (36% vs 14%, $p < 0.001$; Table 3, Fig. 1). The odds of anti-TPO positivity were more than three times higher in the goiter group (OR 3.46, 95% CI 1.72–6.94). Anti-TG positivity was similar in the two groups (15% vs 16%, $p = 0.845$; OR 0.93, 95% CI 0.43–1.99). TRAb positivity was observed only in the goiter group (5% vs 0%), a difference that did not reach statistical significance ($p < 0.059$); an odds ratio could not be estimated because no participant without goiter was TRAb-positive. Overall, at least one antibody was present in 43 of 100 participants with goiter and 20 of 100 participants without goiter (OR 3.02, 95% CI 1.61–5.67). Conversely, 64% of participants with goiter were anti-TPO-negative and 57% were negative for all three antibodies.

Table 3
Frequency of thyroid autoantibody positivity in euthyroid adults with and without goiter

Antibody	Goiter (n = 100) n (%)	No goiter (n = 100) n (%)	Unadjusted OR (95% CI)	p value
Anti-TPO Ab	36 (36.0)	14 (14.0)	3.46 (1.72–6.94)	< 0.001
Anti-TG Ab	15 (15.0)	16 (16.0)	0.93 (0.43–1.99)	0.845
TRAb	5 (5.0)	0 (0.0)	Not estimable	0.059*
Any antibody†	43 (43.0)	20 (20.0)	3.02 (1.61–5.67)	< 0.001

Chi-square test; *Fisher's exact test. †Derived from Table 4 (100 minus the number with no antibody). OR, odds ratio; CI, confidence interval.

Patterns of antibody positivity

The overall distribution of antibody patterns differed significantly between groups ($p < 0.001$; Table 4, Fig. 2). Absence of any antibody was more common in participants without goiter (80.0%) than in those with goiter (57.0%). Isolated anti-TPO positivity was markedly more frequent in the goiter group (24.0% vs 4.0%). Isolated TRAb positivity (2.0%) and combined TRAb-related positivity (TRAb + anti-TPO, 2.0%; TRAb + anti-TG, 1.0%) occurred only in participants with goiter, at low frequency. Isolated anti-TG positivity (4.0% vs 6.0%) and combined anti-TPO + anti-TG positivity (10.0% in both groups) were similar.

Table 4
Patterns of thyroid autoantibody positivity in euthyroid adults with and without goiter

Antibody positivity pattern	Goiter (n = 100) n (%)	No goiter (n = 100) n (%)
No antibody positive	57 (57.0)	80 (80.0)
TRAb only	2 (2.0)	0 (0.0)
Anti-TPO Ab only	24 (24.0)	4 (4.0)
Anti-TG Ab only	4 (4.0)	6 (6.0)
TRAb + Anti-TPO Ab	2 (2.0)	0 (0.0)
TRAb + Anti-TG Ab	1 (1.0)	0 (0.0)
Anti-TPO Ab + Anti-TG Ab	10 (10.0)	10 (10.0)
Overall comparison: exact (Monte Carlo) test, $p < 0.001$.		
.		

Discussion

Principal findings

In this comparative cross-sectional study of 200 euthyroid adults, anti-TPO positivity was more than three times as likely in participants with goiter as in those without (36% vs 14%), and anti-TPO titres were higher. Anti-TG positivity was almost identical in the two groups (15% vs 16%), and TRAb positivity was uncommon and confined to the goiter group (5% vs 0%; p = 0.059). These findings suggest that anti-TPO positivity, a marker of autoimmune thyroiditis, is more common in enlarged than in non-enlarged thyroid glands that are biochemically normal, in a country where iodine deficiency has been substantially reduced by salt iodization [1, 3]. However, most participants with goiter were anti-TPO-negative (64%) and 57% had none of the three antibodies, so autoimmunity, as captured by these markers, does not account for most euthyroid goiters in this sample.

Comparison with previous studies

The anti-TPO positivity in our goiter group is close to the 35% reported in patients with goiter in Nigeria [24] and to the 34–39% reported in Bangladeshi patients with nodular goiter [21]. Hasanat et al., using an antimicrosomal antibody assay, found strong positivity in 19.4% and weak positivity in 7.3% of Bangladeshi patients with thyroid disease, compared with 4.3% and 2.1% in healthy controls [20]. The higher positivity in our comparison group (14% for anti-TPO) may reflect differences in assay and cut-off or in how the group was selected, and is comparable to population-based estimates from iodine-sufficient countries [25].

Anti-TG positivity did not distinguish participants with goiter from those without. This is consistent with reports that anti-TG is less sensitive than anti-TPO for autoimmune thyroid disease, including a South Indian series in which the sensitivity of anti-TPO was 98.1% compared with 61.8% for anti-TG [26, 27]. In contrast, Emran et al. found anti-TG positivity in 27–33% of patients with nodular goiter [21], so the contribution of anti-TG may differ by goiter type. We did not stratify by sonographic pattern.

TRAb was detected in five participants with goiter and in none without. The study was not powered for this outcome, and the difference was not statistically significant. Shefin et al. also observed TRAb in structurally altered thyroid glands without overt hyperthyroidism [28]. Binding assays for TRAb do not distinguish stimulating from blocking antibodies [29], so a positive result in a euthyroid person may represent early subclinical Graves' disease, a blocking antibody, or a low-titre non-functional antibody. Follow-up would be needed to establish its significance.

Demographic and other differences between groups

The goiter group was older, more often female and more often had a family history of thyroid disease. The female predominance (80%) is in keeping with a female-to-male ratio of 7.3:1 in patients with goiter in Nigeria [24] and with the association of female sex with thyroid autoantibody positivity [28, 30]. Plausible explanations include sex-hormone modulation of immune responses and the general predisposition of women to autoimmune disease [31]. A family history of thyroid disease was reported by a quarter of participants with goiter and by none of those without, in line with the strong heritable

component of autoimmune thyroid disease [32, 33]. That no participant in the comparison group reported a family history or diabetes suggests that this group may have been selected as particularly healthy, which could exaggerate contrasts between groups. Because antibody prevalence rises with age and is higher in women [25], confounding by age and sex may account for part of the anti-TPO association, and this cannot be excluded without adjusted analysis.

Overweight and obesity were more frequent in the comparison group than in the goiter group, in contrast to the association between higher BMI and thyroid nodules or larger thyroid volume reported by Dauksiene et al. [34]. Given the non-random selection of the comparison group, we do not interpret this as evidence against a relationship between BMI and goiter.

TSH did not differ between groups, but FT4 was lower in the goiter group (median 1.09 vs 1.31 ng/dL) and TSH tended to be higher (2.75 vs 2.28 μIU/mL), with all values within the reference range. Small shifts of this kind could be compatible with reduced thyroid functional reserve in some glands, since autoantibodies can precede overt dysfunction [19], but the differences are small and their clinical relevance is uncertain. Structural enlargement can also occur without functional change [35].

Implications

If confirmed in larger samples, measuring anti-TPO in euthyroid adults with goiter may help identify people who should be followed for progression to hypothyroidism, as antibody-positive individuals are at higher risk in population studies [19]. Our design cannot assess predictive value or whether testing changes outcomes. Iodine status was not measured; both iodine deficiency and excess have been linked to goiter and to thyroid autoimmunity [17, 18], and residual goiter after iodization has been attributed to iodine status, thiocyanate exposure and autoimmunity [15]. Any interpretation in terms of a post-iodization shift toward autoimmune goiter is therefore hypothetical.

Strengths and limitations

Strengths include the simultaneous measurement of three thyroid autoantibodies on a single platform, biochemical confirmation of euthyroidism in both groups, ultrasonographic assessment by a single sonologist, and the inclusion of a comparison group. The study also has several limitations. First, the cross-sectional design cannot establish temporal sequence or causality. Second, participants were enrolled at a single tertiary-care centre using a non-probability approach, which limits generalisability to the community. Third, the groups differed in age, sex, family history, diabetes and BMI, and no multivariable analysis was performed, so the results are unadjusted associations. Fourth, the comparison group was probably not representative of the general euthyroid population. Fifth, iodine status (for example, urinary iodine concentration) was not assessed, so residual confounding by iodine intake cannot be excluded. Sixth, inter- and intra-observer reliability of ultrasonography was not assessed, and smoking, an established modifier of autoimmune thyroid disease risk [36], was not recorded. Seventh, only 5 participants were TRAb-positive, so the analysis of TRAb has little power. Finally, the reasons for exclusion of the 50 individuals who were screened but not included were not systematically retained, which limits assessment of selection bias.

Conclusions

In this single-centre comparison, anti-TPO positivity was more frequent in euthyroid adults with goiter than in euthyroid adults without goiter, whereas anti-TG and TRAb positivity did not differ significantly. The association was unadjusted, and most participants with goiter had no detectable thyroid autoantibody. Larger multicentre studies that adjust for age, sex, family history and iodine status, and longitudinal studies to determine whether antibody-positive euthyroid goiter progresses to thyroid dysfunction, are needed before anti-TPO testing can be recommended in this group.

Abbreviations

BMI
body mass index
CI
confidence interval
FT4
free thyroxine
IQR
interquartile range
NINMAS
National Institute of Nuclear Medicine and Allied Sciences
OR
odds ratio
SD
standard deviation
TG
thyroglobulin
TPO
thyroid peroxidase
TRAb
thyrotropin receptor antibody
TSH
thyroid-stimulating hormone
WHO
World Health Organization.

Declarations

Ethics approval and consent to participate: The study was approved by the Institutional Review Board of Bangladesh Medical University [approval reference number and date to be inserted] and was conducted

in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants.

Consent for publication: Not applicable.

Availability of data and materials: The datasets generated and analysed during the current study are available from the corresponding author on reasonable request.

Competing interests: The authors declare that they have no competing interests.

Funding: This research did not receive any specific grant from funding agencies in the public, commercial or not-for-profit sectors.

Authors' contributions: SPK, SS, AH, KA, FU, MMR): conception and design, data acquisition, analysis and interpretation, drafting and revision. All authors must read and approve the final manuscript.

Acknowledgements:

This study was acknowledging all the participants who were willingly participants in this study. This study also acknowledge Department of Endocrinlogy, Bangladesh Medical University, Bangladesh.

Clinical trial number: Not applicable.

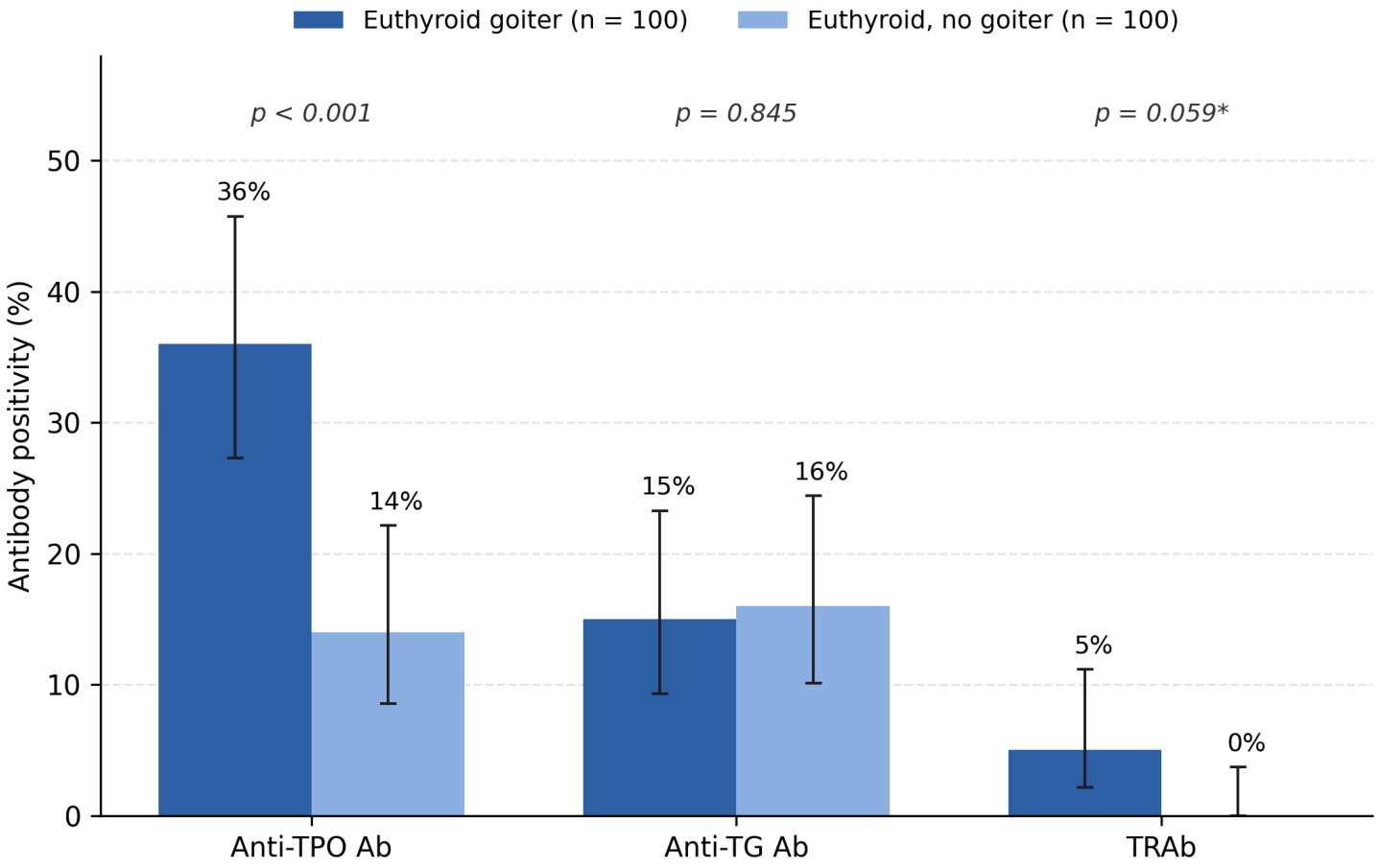
References

1. Yusuf HKM, Quazi S, Khan MR, Mohiduzzaman M, Nahar B, Rahman MM, et al. Iodine deficiency disorders in Bangladesh. Indian J Pediatr. 1996;63(1):105–10.
2. World Health Organization, United Nations Children's Fund, International Council for Control of Iodine Deficiency Disorders. Indicators for assessing iodine deficiency disorders and their control through salt iodization. Geneva: World Health Organization; 1994. WHO/NUT/94.6.
3. Yusuf HKM, Rahman AKMM, Chowdhury FP, Mohiduzzaman M, Banu CP, Sattar MA, et al. Iodine deficiency disorders in Bangladesh, 2004–05: ten years of iodized salt intervention brings remarkable achievement in lowering goitre and iodine deficiency among children and women. Asia Pac J Clin Nutr. 2008;17(4):620–8.
4. Taylor PN, Albrecht D, Scholz A, Gutierrez-Buey G, Lazarus JH, Dayan CM, et al. Global epidemiology of hyperthyroidism and hypothyroidism. Nat Rev Endocrinol. 2018;14(5):301–16.
5. McGrogan A, Seaman HE, Wright JW, de Vries CS. The incidence of autoimmune thyroid disease: a systematic review of the literature. Clin Endocrinol (Oxf). 2008;69(5):687–96.
6. Golden SH, Robinson KA, Saldanha I, Anton B, Ladenson PW. Clinical review: prevalence and incidence of endocrine and metabolic disorders in the United States: a comprehensive review. J Clin Endocrinol Metab. 2009;94(6):1853–78.
7. Vanderpump MPJ. The epidemiology of thyroid disease. Br Med Bull. 2011;99:39–51.

8. Canaris GJ, Manowitz NR, Mayor G, Ridgway EC. The Colorado thyroid disease prevalence study. Arch Intern Med. 2000;160(4):526–34.
9. Caturegli P, Kimura H, Rocchi R, Rose NR. Autoimmune thyroid diseases. Curr Opin Rheumatol. 2007;19(1):44–8.
10. Selim S. Thyroid research in Bangladesh – current status and future directions. Bangladesh J Endocrinol Metab. 2025;4(1):1–3.
11. McLeod DSA, Cooper DS. The incidence and prevalence of thyroid autoimmunity. Endocrine. 2012;42(2):252–65.
12. Führer D, Bockisch A, Schmid KW. Euthyroid goiter with and without nodules—diagnosis and treatment. Dtsch Arztebl Int. 2012;109(29–30):506–15.
13. Caturegli P, De Remigis A, Rose NR. Hashimoto thyroiditis: clinical and diagnostic criteria. Autoimmun Rev. 2014;13(4–5):391–7.
14. Caturegli P, De Remigis A, Chuang K, Dembele M, Iwama A, Iwama S. Hashimoto's thyroiditis: celebrating the centennial through the lens of the Johns Hopkins hospital surgical pathology records. Thyroid. 2013;23(2):142–50.
15. Marwaha RK, Tandon N, Gupta N, Karak AK, Verma K, Kochupillai N. Residual goiter in the postiodization phase: iodine status, thiocyanate exposure and autoimmunity. Clin Endocrinol (Oxf). 2003;59(6):672–81.
16. Jayatissa R, Okosieme OE, Ranasinghe S, Carter JL, Gunatunga IP, Lazarus JH, et al. Thyroid autoimmunity and dysfunction in Sri Lankan children and adolescents after 22 years of sustained universal salt iodization. Thyroid. 2021;31(7):1105–13.
17. Teng W, Shan Z, Teng X, Guan H, Li Y, Teng D, et al. Effect of iodine intake on thyroid diseases in China. N Engl J Med. 2006;354(26):2783–93.
18. Zimmermann MB, Boelaert K. Iodine deficiency and thyroid disorders. Lancet Diabetes Endocrinol. 2015;3(4):286–95.
19. Vanderpump MPJ, Tunbridge WMG, French JM, Appleton D, Bates D, Clark F, et al. The incidence of thyroid disorders in the community: a twenty-year follow-up of the Whickham Survey. Clin Endocrinol (Oxf). 1995;43(1):55–68.
20. Hasanat MA, Rumi MA, Alam MN, Hasan KN, Salimullah M, Salam MA, et al. Status of antithyroid antibodies in Bangladesh. Postgrad Med J. 2000;76(896):345–9.
21. Emran MS, Hasanat MA, Hassan MQ, Saleh AA, Khan MA, Hossain A, et al. Increased frequency of positive antithyroid antibody in nodular goiter. Bangabandhu Sheikh Mujib Med Univ J. 2016;8(2):109–13.
22. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. Lancet. 2007;370(9596):1453–7.

23. Guth S, Theune U, Aberle J, Galach A, Bamberger CM. Very high prevalence of thyroid nodules detected by high frequency (13 MHz) ultrasound examination. Eur J Clin Invest. 2009;39(8):699–706.
24. Ojo O, Ikem R, Kolawole B, Ojo O, Ajala M. Prevalence and clinical relevance of thyroid autoantibodies in patients with goitre in Nigeria. J Endocrinol Metab Diabetes S Afr. 2019;24(3):92–7.
25. Hollowell JG, Staehling NW, Flanders WD, Hannon WH, Gunter EW, Spencer CA, et al. Serum TSH, T4, and thyroid antibodies in the United States population (1988 to 1994): National Health and Nutrition Examination Survey (NHANES III). J Clin Endocrinol Metab. 2002;87(2):489–99.
26. Jantikar AM. A study on relationship between thyroid peroxidase antibodies (Anti-TPO antibodies) and thyroid dysfunction patients. Int J Clin Biochem Res. 2020;7(2):238–42.
27. Shinto AS, Pachen L, Sreekanth TK, George D. Prevalence of antithyroid antibodies in histologically proven autoimmune thyroid diseases and correlation with thyroid dysfunction in South India. [journal name be added]. 2010;5(9):1–5.
28. Shefin SM, Sobhan R, Ibrahim MI, Islam S. Association between thyroid autoantibodies and abnormal thyroid function and structure of thyroid gland. Sch J Appl Med Sci. 2023;11(11):1903–12.
29. Ross DS, Burch HB, Cooper DS, Greenlee MC, Laurberg P, Maia AL, et al. 2016 American Thyroid Association guidelines for diagnosis and management of hyperthyroidism and other causes of thyrotoxicosis. Thyroid. 2016;26(10):1343–421.
30. Yan YR, Gao XL, Zeng J, Liu Y, Lv QG, Jiang J, et al. The association between thyroid autoantibodies in serum and abnormal function and structure of the thyroid. J Int Med Res. 2015;43(3):412–23.
31. Ngo ST, Steyn FJ, McCombe PA. Gender differences in autoimmune disease. Front Neuroendocrinol. 2014;35(3):347–69.
32. Brix TH, Kyvik KO, Christensen K, Hegedüs L. Evidence for a major role of heredity in Graves' disease: a population-based study of two Danish twin cohorts. J Clin Endocrinol Metab. 2001;86(2):930–4.
33. Antonelli A, Ferrari SM, Corrado A, Di Domenicantonio A, Fallahi P. Autoimmune thyroid disorders. Autoimmun Rev. 2015;14(2):174–80.
34. Dauksiene D, Petkeviciene J, Klumbiene J, Verkauskiene R, Vainikonyte-Kristapone J, Seibokaite A, et al. Factors associated with the prevalence of thyroid nodules and goiter in middle-aged euthyroid subjects. Int J Endocrinol. 2017;2017:1–8.
35. Cooper DS, Biondi B. Subclinical thyroid disease. Lancet. 2012;379(9821):1142–54.
36. Wiersinga WM. Smoking and thyroid. Clin Endocrinol (Oxf). 2013;78(2):145–53.

Figures



Error bars: Wilson 95% CI. p-values: chi-square test (anti-TPO, anti-TG); *Fisher's exact test (TRAb).

Figure 1

Frequency of anti-TPO, anti-TG and TRAb positivity in euthyroid adults with goiter (n = 100) and without goiter (n = 100). Anti-TPO positivity was more frequent in the goiter group (36% vs 14%, p < 0.001), whereas anti-TG (15% vs 16%, p = 0.845) and TRAb (5% vs 0%, p = 0.059) positivity did not differ significantly. Positivity was defined as anti-TPO > 30 IU/mL, anti-TG > 95 IU/mL and TRAb > 1.5 IU/L. Anti-TPO, anti-thyroid peroxidase antibody; anti-TG, anti-thyroglobulin antibody; TRAb, thyrotropin receptor antibody

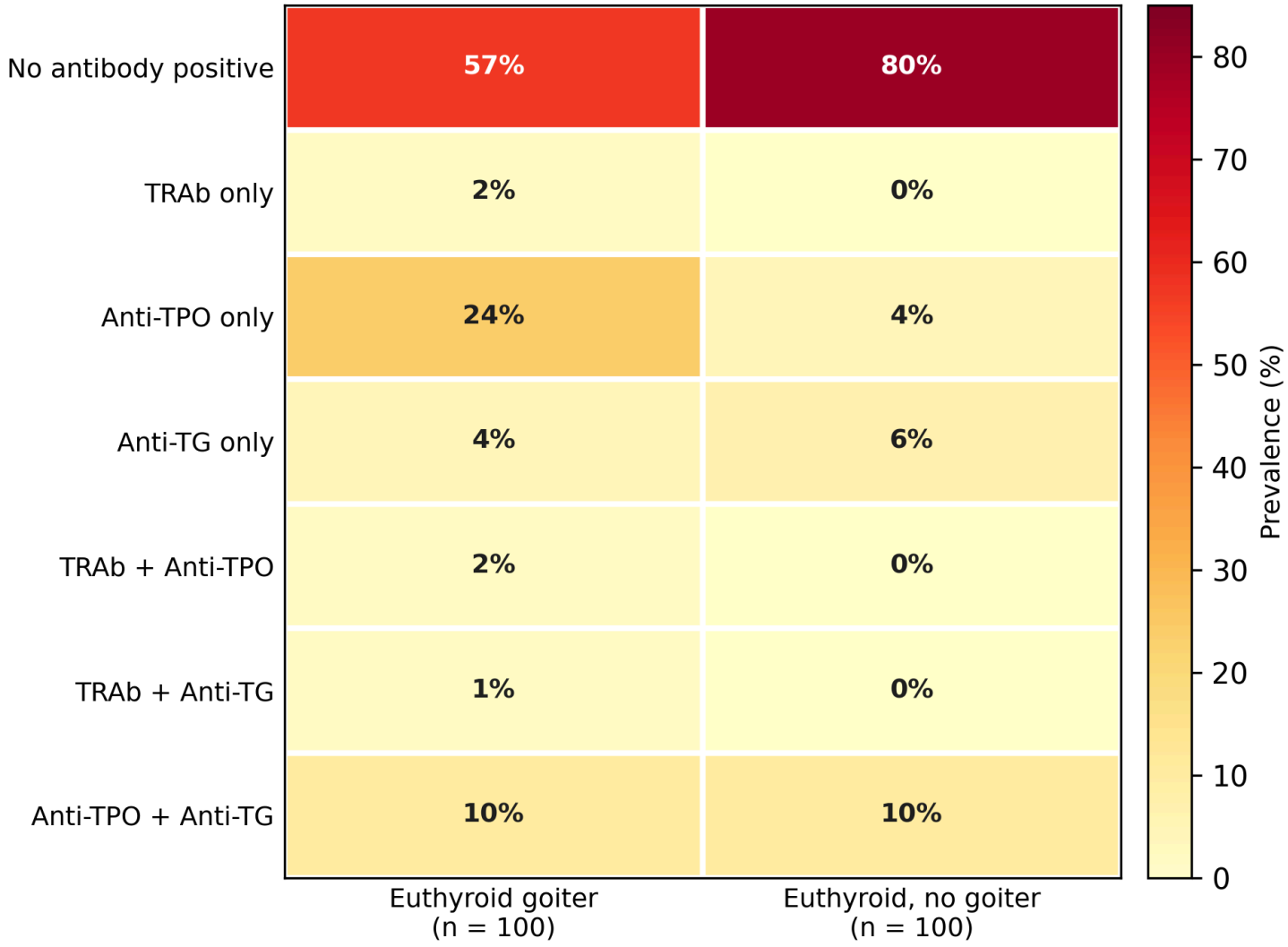


Figure 2

Patterns of thyroid autoantibody positivity in euthyroid adults with goiter (n = 100) and without goiter (n = 100). Proportions of participants negative for all three antibodies (57% vs 80%) or positive for isolated anti-TPO (24% vs 4%), isolated anti-TG (4% vs 6%), anti-TPO + anti-TG (10% vs 10%), isolated TRAb (2% vs 0%), TRAb + anti-TPO (2% vs 0%) and TRAb + anti-TG (1% vs 0%). The overall distribution differed between groups ($p < 0.001$, exact Monte Carlo test). Anti-TPO, anti-thyroid peroxidase antibody; anti-TG, anti-thyroglobulin antibody; TRAb, thyrotropin receptor antibody