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Analytical evaluation of anti-thyroid peroxidase II assay on Atellica IM analyser: what to expect when changing methods

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Abstract

Objectives: The anti-TPO assays are known for the method variability. The new aTPOII assay (Siemens Healthineers) has a different TPO antigen and detecting antibody source and it is also traceable to another international standard compared to the old aTPO assay (Siemens Healthineers). The aims of this study were to verify the new assay through a method comparison (n=860) with the old aTPO assay from the same manufacturer and to verify the reference range (n=60) on Atellica Immunoanalyzer (Atellica IM).

Methods: A total of 860 patient serum samples were measured using the Atellica IM aTPO and Atellica IM aTPOII assays and the positive percent agreement, negative percent agreement, and overall percent agreement (OPA) were calculated along with their corresponding 95 % confidence interval (CI). The reference interval verification on 60 euthyroid patient samples was done.

Results: In view of the substandard quantitative agreement between methods, a qualitative assay comparison was performed and demonstrated that there was good sensitivity (81.5 %; 95 % CI: 77.1–85.3 %), specificity (99.6 %; 95 % CI: 98.6–99.9 %) and OPA (92.6 %; 95 % CI: 90.6–94.1 %). The computed Cohen kappa was 0.84 (95 % CI: 0.80–0.88) which reflects a very good strength of qualitative agreement. The manufacturer's reference interval (cut-off ≤13.8 U/mL) was verified and confirmed.

Conclusions: Using the new aTPOII assay more patients will have negative results. Even though assays claim to be referenced to the corresponding WHO International reference preparation, this standardization does not ensure cut-offs and/or results that are identical and does not guarantee method agreement and assays could not be used interchangeably.

Keywords: anti-thyroid peroxidase; standardization; method comparison; verification; reference intervals

Introduction

Thyroid peroxidase (TPO) is a membrane-bound, glycosylated, haem-containing protein located at the apical membrane of thyroid follicular cells. It constitutes the principal component of the thyroid microsomal antigen and catalyses the iodination of tyrosyl groups in thyroglobulin, resulting in the synthesis of the thyroid hormones T3 and T4 [1]. Measurement of anti–thyroid peroxidase (anti-TPO) antibodies is essential for the diagnosis of autoimmune thyroid disease (AITD), among which the two main diseases are the Hashimoto thyroiditis (HT) and the Graves' disease (GD) [2, 3]. The overall prevalence of AITDs is estimated at approximately 5 %, whereas the prevalence of anti-TPO antibodies in the general population ranges between 10 % and 20 % [4]. Nevertheless, the diagnostic significance of the anti-TPO tests remains paramount, since the prevalence of anti-TPO antibodies among AITD patients ranges from 90 % to 95 % in cases of HT and up to 70–80 % in active GD [3, 5, 6]. Consequently, the presence or absence of these antibodies serves as an effective diagnostic aid, yet their interpretation must always be interpreted in conjunction with clinical findings and other indicators.

It is well established that the autoimmune response to TPO mediated by anti-TPO antibodies is closely associated

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with a multitude of different epitopes on TPO molecule including the enzymatic site of the TPO [1]. This phenomenon also explains the low within-subject biological variation and the high between-subject biological variation observed for thyroid autoantibodies [1, 7]. The anti-TPO assays are known for the method depended variability, largely due to antibody heterogeneity inherent in the patients, which cannot be influenced by standardisation efforts [8]. Thus, given the recognised variability and inadequate standardisation amongst the methods, it is imperative to evaluate both the analytical and clinical performance of commercially available thyroid autoantibody assays prior to their routine implementation.

Recently, Siemens Healthineers announced the availability of the new Atellica IM Anti-Thyroid Peroxidase II (aTPOII) assay for use on the Atellica IM Analyzer [9]. The information provided offered a concise overview of the key features (namely, the new assay structure, additional standardisation, and enhanced analytical performance), but lacked detailed elaboration. In particular, no quantitative method comparison was provided comparing the new Atellica IM aTPOII and the old Atellica IM aTPO assay [10]. Instead, it was stated that results, obtained using different anti-TPO assay methods cannot be used interchangeably.

The objectives of the present study were twofold: firstly, to compare the novel aTPOII assay with the previous aTPO assay from the same manufacturer on the Atellica IM Analyzer over a longer period, with the purpose of providing clinicians with insights into how patient values change depending on the used assay; and secondly, to verify the reference range of the new aTPOII assay.

Materials and methods

Subjects

This prospective study was conducted at the Institute for Clinical Chemistry and Pathobiochemistry at the University Hospital of Tübingen from January until August 2025. Blood samples from in and outpatients with request for thyroid antibody testing were collected in serum tubes (S-Monovette, Serum CAT 4.0 mL, Sarstedt, Nümbrecht, Germany). Residual, anonymised material was then utilised for the purpose of comparison of two thyroid peroxidase antibody assays: the anti-thyroid peroxidase (aTPO) vs. the anti-thyroid peroxidase II (aTPOII, both from Siemens Healthineers, Eschborn, Germany). The inclusion criteria encompassed all in and outpatients for whom a primary aTPO test was requested, irrespective of their age, gender or the nature of their illness. Patients for whom it was not possible to perform both tests on the same sample due to insufficient volume, or on the same day, were excluded from the study. Further stratification based on clinical diagnosis was not performed. All patients provided written informed consent. Approval for the study was granted by the local Ethics Committee of the Medical Faculty and University Hospital of Tübingen, Germany (Nr. 512/2025A).

The reference range verification process involved the selection of an apparently healthy population. This population sample was drawn from a cohort undergoing general antibody testing. Cohort selection was based on the completion of a questionnaire assessing the individuals' medical history including medication use and the presence of chronic diseases such as diabetes mellitus, autoimmune diseases, allergies, chronic kidney disease, and coronary disease. The absence of acute inflammation was determined by clinical assessment conducted by a physician. It is important to note that no physical examinations (e.g. thyroid ultrasound or palpation) were performed in this population.

Methods

Following clot formation, blood samples were centrifugated at 2,700*g for 7 min using a Rotanta 460 robotic swing-out rotor centrifuge (Hettich, Tuttlingen, Germany). Sera were measured within 2 h on an Atellica Solution IM Analyzer (Siemens Healthineers, Eschborn, Germany) using booth assays. The new aTPOII assay was evaluated for precision and accuracy using commercially available quality control (QC) material (Inteliq Specialty Immunoassay Control, BioRad, Feldkirchen, Germany) in accordance with the Clinical and Laboratory Standards Institute's (CLSI) EP15-A3 protocol [11].

A major distinction between the old aTPO and the new aTPOII assay lies in the origin of the TPO antigen (native human vs. recombinant monkey) and the associated traceability to NIBSC 66/387 and WHO 19/260 [12].

Statistical analysis

Precision (between-run and within-laboratory) and bias were determined in accordance with CLSI Document EP15-A3 (basic 5 × 5 design). Two samples with different measurand concentrations were assayed over a period of five days, with one run per day and five replicates per run. In addition, long-term within-laboratory precision was evaluated at the end of the study prior to introducing the assay into routine use.

For quantitative comparability of the old aTPO and the new aTPOII assay, Pearson correlation coefficient (r),

Passing-Bablok regression and Bland-Altman analyses were performed. In order to ensure the comparability of the qualitative data, the numerical data were transformed into binary data and labelled „negative" (within the reference range) or "positive" (above the upper limit of the reference range). Specifically, the reference range for aTPO negative is ≤60 IU/mL, and the reference range for aTPOII negative is ≤13.8 IU/mL and qualitative assay comparison was performed using a 2 × 2 contingency table in accordance with the CLSI document EP12-A2 [13].

In this study, concordant pairs were defined as those consisting of negative/negative and positive/positive combinations, while discordant pairs were defined as those consisting of negative/positive and positive/negative combinations. The positive percent agreement (PPA), negative percent agreement (NPA), and overall percent agreement (OPA), along with their corresponding 95 % confidence intervals (CI), were calculated. Also, Cohen's kappa coefficient for inter-rater agreement with linear weights was utilised.

The reference range verification was performed in accordance with the recommendations set out in CLSI Document EP28-A3c [14].

All statistical analyses were conducted utilising the Microsoft Excel 2010 add-in Analyse-it (Method Validation edition; Analyse-it for Microsoft Excel 5.11) or the statistical software R (Version R-4.5.1, R Core Team, USA).

Results

Performance characteristics

The results presented in Tables 1 and 2 are indicative of the assay's performance. The results obtained showed great performance with a coefficient of variation (within-laboratory precision) below 2.5 % and mean bias below 5.5 %. Throughout the study duration, long-term imprecision for both control levels remained below 10 % (control level 1: 8.7 %; control level 2: 6.0 %).

Table 1: Precision of the aTPOII assay on the Atellica IM analyser using the basic 5 × 5 design.

Sample	Mean, IU/mL	Between run		Within laboratory	
		SD, IU/mL	CV, %	SD, IU/mL	CV, %
Control level 1	25.6	0.42	1.65	0.58	2.25
Control level 2	74.2	1.11	1.50	1.68	2.26

SD, standard deviation; CV, coefficient of variation. Control level 1 – InteliQ specialty immunoassay control (lot: 10401T). Control level 2 – InteliQ specialty immunoassay control (lot: 10402T).

Table 2: Bias of the aTPOII assay on the Atellica IM analyser.

Sample	Mean, IU/mL	Bias	
		IU/mL	%
Control level 1	25.6	1.45	5.36
Control level 2	74.2	2.6	3.4

Control level 1 – InteliQ specialty immunoassay control (lot: 10401T).
Control level 2 – InteliQ specialty immunoassay control (lot: 10402T).

Method comparison

During the evaluation period, a total of 1,594 requests for anti-TPO measurements were received. According to the exclusion criteria 734 patients were excluded from the study, leaving 860 sera available for method comparison. The obtained variation as determined by regression analysis (y=5.053+0.04745×; 95 % CI for intercept: 4.285–6.099 and for slope: 0.01556–0.07213) was indicative of the differences between the quantitative results (Figure 1A). In Figure 1B, the differences between two assays are shown in percentages. Mean difference of 135 % confirmed that these two assays do not agree quantitatively. The correlation coefficient (r=0.776) generally indicates poor correlation. In view of the substandard quantitative agreement between methods, a qualitative assay comparison was performed demonstrating good specificity, sensitivity and overall percent agreement (see Table 3). The computed Cohen kappa was 0.84 (95 % CI: 0.80–0.88) which reflects a very good strength of qualitative agreement.

Verification of reference interval

The verification process was initiated with a sample of 20 individuals. However, three individuals in the initial group exhibited values exceeding the predefined cut-off threshold, necessitating repetition of the procedure. Consequently, a full cohort of 60 individuals aged 18–65 years was used, with a balanced proportion of males and females. Further measurements of TSH (normal range: 0.5–4.4 mU/L) and thyroid hormones (normal ranges: fT3: 3.5–6.5 pmol/L; fT4: 11–23 pmol/L) were obtained from this cohort to confirm euthyroid status. The application of the Reed–Dixon method resulted in the identification of outliers, with a maximum of 5 % of results (n=3) falling outside the reference cut-off. The study revealed that two of 60 individuals exhibited values

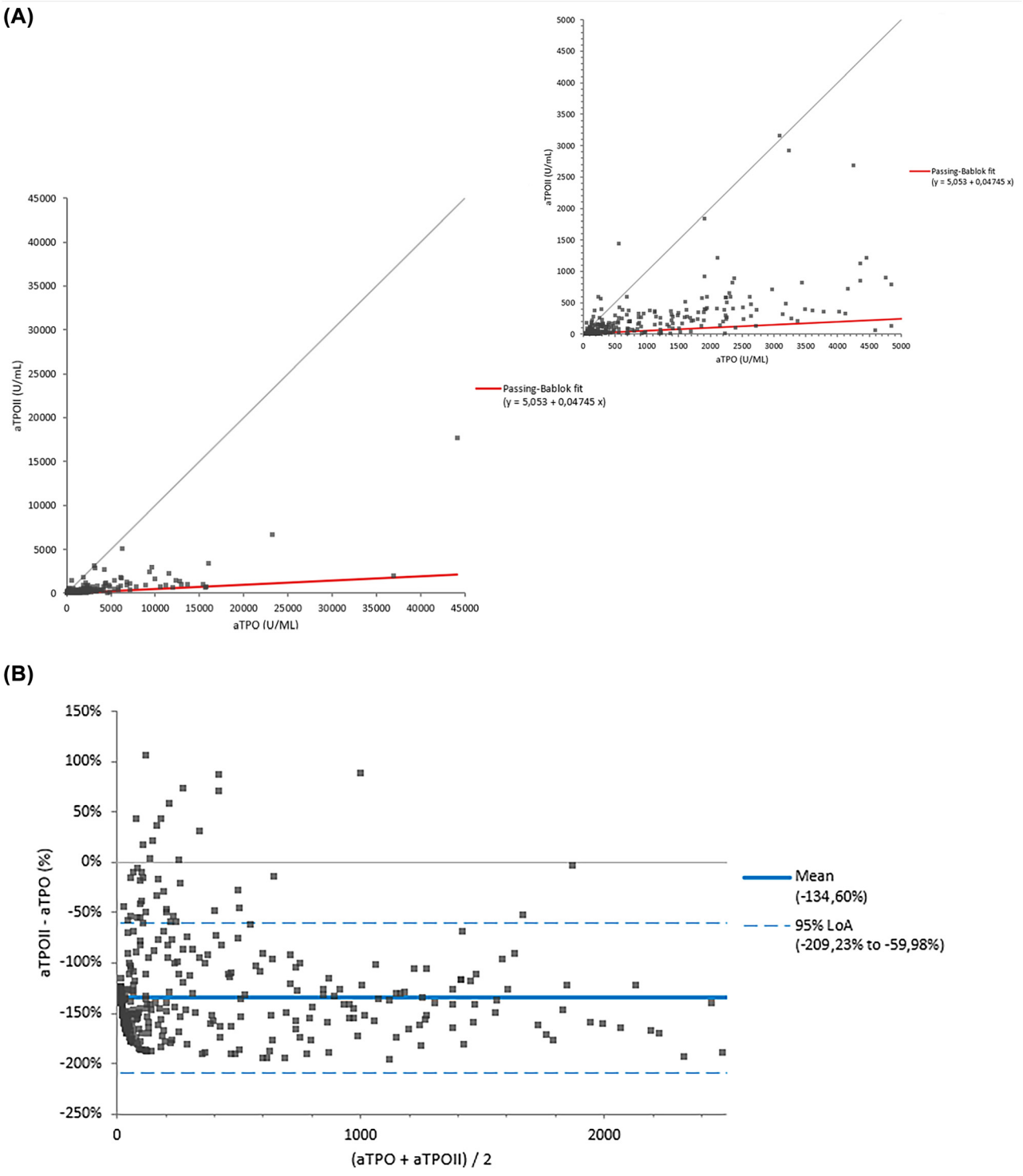

Figure 1: (A) Passing-Bablok regression analysis with included magnified, low-concentration graph as an insert showing the comparison of anti-TPO measurements between the aTPO and the aTPOII on the Atellica IM analyser. Red line represents Passing-Bablok fit. (B) Bland-Altman plot showing the mean bias (relative differences) of anti-TPO measurements between the aTPO and the aTPOII on the Atellica IM analyser.

above the limit of quantification, yet below the cut-off point. In contrast, 55 of 60 individuals demonstrated values below the limit of quantification. The results of the study confirmed the manufacturer's cut-off value of ≤13.8 U/mL.

Table 3: Assay comparison.

		Atellica IM aTPO assay		
		≥60 U/mL	<60 U/mL	Total
Atellica IM aTPOII assay	≥13.8 U/mL	274	2	276
	<13.8 U/mL	62	522	584
	Total	336	524	860

aTPOII, anti-thyroid peroxidase II; aTPO, anti-thyroid peroxidase. Positive percent agreement: 81.5 % [95 % confidence interval: 77.1–85.3 %]. Negative percent agreement: 99.6 % [95 % confidence interval: 98.6–99.9 %]. Overall percent agreement: 92.6 % [95 % confidence interval: 90.6–94.1 %].

Discussion

To ensure the reliability of the results obtained, a verification procedure is essential. The process under discussion involves the assessment of assay repeatability, reproducibility, sensitivity, specificity, and reference intervals, with the aim to evaluate overall performance. The aforementioned performances are conducted in accordance with quality standards, including those defined by the international guidelines such as International Organization for Standardization (ISO 15189). We evaluated the performance of the new aTPOII assay on the Atellica IM prior to its routine implementation, comparing it with the previous aTPO assay.

The new assay has demonstrated acceptable within-laboratory and between-run imprecision, with even lower CV for both QC levels than those specified in the package insert, confirming the claims that the novel assay exhibits improved analytical performance compared with the previous assay [9, 10]. Notwithstanding the great precision of the assay, it is noteworthy that both QC levels are indicative of pathological values, suggesting potential limitations in analytical performance near the decision thresholds of 13.8 U/mL. The manufacturer provided data indicating analytical performance of <15 % for concentrations <17 U/mL [12]. This claim can also be relevant, since the low calibrator is approximately 22.5 IU/mL, with a measurement uncertainty of 4.3 %. To evaluate this analytically critical aspect, the analytical performance at 13.6 U/mL by diluting control material level 1 with sample diluent at a ratio of 1:2, and by calculating imprecision using the same basic 5×5 design was assessed. Furthermore, our data showed that even within this low concentration range, the assay demonstrated great performance, with a within laboratory precision of 4.3 % and an expanded measurement uncertainty of 12.2 % which is acceptable compared to our maximum allowable expanded uncertainty of 24.5 %.

It has been reported by several authors that there is poor correlation between different anti-TPO assays [8, 15, 16]. In our study, even if assays from the same manufacturer were compared, we obtained results similar to those previously published for assays from different manufacturers [8, 15, 16]. The observed discrepancy may be attributable to assay standardisation. Specifically, the aTPO assay exhibited traceability to the theoretical WHO International Units through a conversation factor that was, on average, three-fold higher (ranging from two- to four-fold across the assay range). The manufacturer asserts that the novel aTPOII assay is directly standardised to NIBSC 66/387 and traceable to new WHO standard 19/260. However, ss WHO 19/260 is not commutable with the NIBSC 66/387 standard, exact equivalency between the two assay is not achievable. However, the aTPOII is designed to trace well to this new standard, with a slope of 1.5. Furthermore, inter-assay variation can also be due to the antigen used, since aTPO employs native human TPO (comprising full-length, transmembrane and intracellular domains) whereas aTPOII utilises recombinant monkey (96 % homologous) TPO antigen (extracellular portion) [12, 17]. Given that the new assay employs recombinant monkey TPO, any potential species-related epitope differences or cross-reactivity should be explicitly evaluated in additional experimental studies. Comparative binding analyses using both human and monkey TPO would clarify whether the antibody targets conserved or species-restricted domains, while epitope mapping or competition assays could provide further insight into the impact of species-specific structural variations on antibody affinity.

In real-world testing conditions, slight variations are expected when comparing any two immunoassays, particularly at clinical decision thresholds. The assay described in the present study align with the manufacturer's assertion that it is imperative for both positive and negative percent agreements to exceed 97 %. This was not a finding that emerged from our study. The PPA of the aforementioned product was found to be 81.5 %, in comparison to the manufacturer's stated claim of 99.2 %. This unsatisfactory outcome can be attributable to differences in the study populations between our study and that of the manufacturer (n=850 in and outpatients in our study vs. 244 outpatients, of whom 142 were healthy, in manufacturer's study). Furthermore, the inclusion of a mixed population of inpatients and outpatients without detailed clinical context may inflate heterogeneity and obscure the interpretation of the PPA. Therefore, we additionally studied the distribution of TSH and free thyroid hormone levels in these individuals, since these levels influence antibody prevalence. The study

revealed that three out of 62 individuals exhibited hypothyroidism (high TSH, low fT3/fT4), and five exhibited hyperthyroidisms (low TSH, high fT3/fT4). In contrast, 54 out of 62 individuals demonstrated values within the euthyroid range. Furthermore, the high negative agreement is of particular importance in the reliable exclusion of disease. Our study demonstrated specificity of 100 % which is in accordance with manufacturer claim of 98.4 %.

In light of the alterations observed in the standardization and reagent composition, the manufacturer's established cut-off of 13.8 U/mL has been verified on a population of 60 healthy euthyroid individuals.

The conduct of comparison studies is of paramount importance for method validation and the provision of optimal patient care. In the event that two methods are demonstrated to be in agreement, they may be declared comparable and consequently employed interchangeably. Irrespective of the outcomes of such comparative studies, it is imperative that physicians are advised in order to enable them to make informed medical decisions [18]. In circumstances when a change in methodology occurs, there is the possibility that previously positive patients may be reclassified as negative, and conversely. Therefore, it is crucial to employ a singular assay method for longitudinal patient monitoring. When transitioning to a new method, a new baseline must be established for all patients.

To the best of our knowledge, this is the first comparison study conducted using the new aTPO II assay, providing insights into a large set of quantitative and qualitative data. The study's main strengths are its extensive evaluation period and the large number of participants, during which for the majority of patients were able to establish a new baseline. This facilitated a seamless transition to a new assay, ensuring that all physicians had a reliable starting point. The communication strategy implemented during the study was instrumental in preventing erroneous interpretations and fostering a collaborative environment. Conversely, the primary constraint pertains to the absence of patient clinical data, a deficiency that hinders the evaluation of clinical sensitivity and specificity. This prerequisite is pivotal for the interpretation of assay accuracy beyond the confines of analytical agreement.

In conclusion, the new aTPOII assay will produce more negative results from an analytical perspective. It is important to note that some autoimmune patients could be reclassified as seronegative. This is crucial for estimating the clinical consequences of changing tests. Even though assays claim to be referenced to the corresponding WHO International Reference Preparation, this standardization does not ensure cutoffs and/or results that are identical and does not guarantee method agreement and assays could not be used interchangeably. In addition, results of the anti-TPO assays should only be used as an aid in diagnosis in conjunction with clinical symptoms and medical history. Therefore, anti-TPO negativity/positivity is not the sole determinant in diagnosing autoimmune thyroiditis.

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Research ethics: Approval for the study was granted by the local ethics committee of the Medical Faculty and University Hospital of Tübingen, Germany (Nr. 512/2025A).
Informed consent: All patients provided written informed consent.
Author contributions: Conceptualization: MK, RK, SH and AP designed the study, developed the theory, and planned the experiments. Data Collection & Investigation: MK, RK and AS performed the laboratory experiments, while TG curated the datasets. Analysis & Visualization: TG and SH applied statistical techniques to analyze the data and created the figures. Writing – Original Draft: MK and AS wrote the initial manuscript draft. Writing – Review & Editing: All authors provided critical feedback, reviewed the results, and approved the final manuscript. Supervision: AP supervised the project. All authors have accepted responsibility for the entire content of this manuscript and approved its submission.
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References

1. Kaczur V, Vereb G, Molnár I, Krajczár G, Kiss E, Farid NR, et al. Effect of anti-thyroid peroxidase (TPO) antibodies on TPO activity measured by chemiluminescence assay. Clin Chem 1997;43:1392–6.
2. Kasagi K, Kousaka T, Higuchi K, Iida Y, Misaki T, Alam MS, et al. Clinical significance of measurements of antithyroid antibodies in the diagnosis of Hashimoto's thyroiditis: comparison with histological findings. Thyroid 1996;6:445–50.

3. Fröhlich E, Wahl R. Thyroid autoimmunity: role of anti-thyroid antibodies in thyroid and extra-thyroidal diseases. Front Immunol 2017; 8:521.
4. Jacobson DL, Gange SJ, Rose NR, Graham NM. Epidemiology and estimated population burden of selected autoimmune diseases in the United States. Clin Immunol Immunopathol 1997;84:223–43.
5. Mehanathan PB, Erusan RR, Shantaraman K, Kannan SM. Antithyroid peroxidase antibodies in multinodular Hashimoto's thyroiditis indicate a variant etiology. J Thyroid Res 2019;2019:4892329.
6. Vargas-Uricoechea H, Nogueira JP, Pinzón-Fernández MV, Schwarzstein D. The usefulness of thyroid antibodies in the diagnostic approach to autoimmune thyroid disease. Antibodies 2023;12:48.
7. González C, Hernando M, Cava F, Herrero E, García-Díez LC, Navajo JA, et al. Biological variability of thyroid autoantibodies (anti-TPO and anti-Tg) in clinically and biochemically stable patients with autoimmune thyroid disease. J Clin Lab Anal 2002;16:37–9.
8. La'ulu SL, Slev PR, Roberts WL. Performance characteristics of 5 automated thyroglobulin autoantibody and thyroid peroxidase autoantibody assays. Clin Chim Acta 2007;376:88–95.
9. Siemens Healthineers. Customer bulletin. Supersedes 11695708, Rev A. Tarrytown, NY, USA: Siemens Healthcare Diagnostics Inc.; 2024.
10. Siemens Healthineers. Customer bulletin. Supersedes 11695527, Rev C. Tarrytown, NY, USA: Siemens Healthcare Diagnostics Inc.; 2025.
11. CLSI. User verification of precision and estimation of bias; approved guideline, 3rd ed. CLSI document EP15-A3. Wayne (PA): Clinical and Laboratory Standards Institute; 2014.
12. Package Insert. Anti-thyreoperoxidase II (aTPOII) 11206083_EN. Gwynedd (UK): Siemens healthcare diagnostics products Ltd. Rev 02 2025.
13. CLSI. User protocol for evaluation of qualitative test performance; approved guideline, 2nd ed. CLSI document EP12-A2. Wayne (PA): Clinical and Laboratory Standards Institute; 2008.
14. CLSI. Defining, establishing and verifying reference intervals in the clinical laboratory; approved guideline. In: CLSI document C28-A3c, 3rd ed. Wayne (PA): Clinical and Laboratory Standards Institute; 2010.
15. Nishihara E, Amino N, Kudo T, Ito M, Fukata S, Nishikawa M, et al. Comparison of thyroglobulin and thyroid peroxidase antibodies measured by five different kits in autoimmune thyroid diseases. Endocr J 2017;64:955–61.
16. Manescu IB, Luca A, Hutanu A, Truta A, Dobrenau M. Anti-thyroid peroxidase (TPO) antibodies: comparative analysis of two automatic methods, ECLIA and CMIA. Rev Rom Med Lab 2024;32:33–40.
17. Package Insert. Anti-thyreoperoxidase (aTPO) 10995280_EN. Tarrytown (NY): Siemens healthcare diagnostics products Ltd. Rev 04 2024.
18. Huang Z, He M, Wang X. Performance verification of Siemens Atellica DL IM1600 automatic immune analyzer detection system: A-TPO and A-TG. Medicine (Baltim) 2024;103:e40168.