RESEARCH ARTICLE

Potential diagnostic role of serum asprosin in Graves' disease with subgroup analysis of subclinical hyperthyroidism: A Case–Control Study

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Background: Asprosin is a recently identified adipokine predominantly released by white adipose tissue and plays a role in gluconeogenesis during fasting. Evidence indicates that adipokines may be involved in endocrine and metabolic control; however, their significance in thyroid dysfunction is still unclear.

Objective: This study aimed to assess circulating asprosin levels in patients with Graves' disease (GD) and subclinical hyperthyroidism (SCH), as well as determine its potential diagnostic use as a biomarker for thyroid disorders.

Methods: This age-, sex-, and BMI-matched case–control study included 40 patients with Graves' disease (GD), 40 with subclinical hyperthyroidism (SCH), and 80 healthy controls aged 22–59 years. Serum levels of asprosin, thyroid-stimulating hormone (TSH), total thyroxine (T4), thyroid-stimulating hormone receptor antibodies (TRAb), and lipid profile parameters were measured using standardized automated assays.

Results: Serum asprosin levels were significantly lower in patients with Graves' disease and subclinical hyperthyroidism compared with healthy controls ($p < 0.001$ for both). Both GD and SCH groups also exhibited significantly reduced levels of TSH and lipid profile parameters, alongside significantly elevated total T4 and TRAb levels ($p < 0.001$).

Conclusion: Reduced serum asprosin in Graves' disease may reflect hyperthyroidism-related metabolic changes and could serve as a potential biomarker, pending further research.

Keywords: asprosin, graves' disease, hyperthyroidism, subclinical hyperthyroidism.

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Introduction

Thyroid hormones play a critical role in regulating metabolism, growth, and development in humans; thyroid disorders, such as hyperthyroidism or hypothyroidism, can impair several critical physiological processes [1]. Graves' disease (GD) is the most common cause of hyperthyroidism and is characterized by autoimmune stimulation of the thyroid gland, leading to excessive production of thyroid hormones. This systemic autoimmune condition impacts multiple organs, primarily the thyroid, eyes (causing Graves' ophthalmopathy), and skin (manifesting resulting in pretibial myxedema) [2]. It primarily occurs between the ages of 30 and 60, with a prevalence 5 to 10 times greater in women. Approximately 79% of the risk for GD is linked to genetic factors, while 21% arises from environmental influences. Approximately 70% of the genes linked to autoimmune thyroid diseases are related to T-cell function [3]. Subclinical hyperthyroidism (SCH) is a disorder characterized by decreased blood levels of thyroid-stimulating hormone (TSH) while maintaining normal circulating levels of free thyroxine (fT_4) and free triiodothyronine (fT_3). It is marked by normal thyroid hormone levels, unlike overt hyperthyroidism, and often exhibits few clinical signs [4].

Asprosin is a newly identified hormone stimulated by fasting, primarily secreted by white adipose tissue. First described in 2016 as the C-terminal cleavage derivative of profibrillin-1, it is encoded by the FBN1 gene located on chromosome 15q21.1. After post-translational modification, circulating human asprosin has an approximated molecular weight of 30 kDa [5].

Asprosin is physiologically vital in regulating energy balance during fasting phases. During its release from adipocytes into the bloodstream, it interacts with the olfactory receptor OLFR734 in the liver, thereby stimulating the G protein–cyclic AMP (cAMP)–protein kinase A (PKA) signaling cascade. This cascade promotes hepatic gluconeogenesis and glycogenolysis, resulting in elevated glucose release into the bloodstream and the maintenance

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of normal glycemic levels during energy deficiency. Besides its peripheral metabolic effects, asprosin passes the blood-brain barrier and activates orexigenic neurons in the hypothalamus, thus increasing appetite and enhancing adaptive responses to fasting [6]. In addition to its physiological function, asprosin has been associated with many metabolic and inflammatory disorders. Increased circulating levels have been related to insulin resistance, obesity, polycystic ovarian syndrome, type 2 diabetes mellitus, and cardiovascular disease [7]. In contrast, asprosin deficiency due to FBN1 mutations has been associated with hypoglycemia and diminished appetite in patients with neonatal progeroid (Wiedemann–Rautenstrauch) syndrome [8].

In hyperthyroid states, regardless of the underlying cause, elevated thyroid hormone levels increase the basal metabolic rate and enhance substrate utilization. This impact is partially mediated by the increase of β-adrenergic receptor density and sensitivity in adipocytes, which enhances catecholamine-stimulated adenylate cyclase activity and results in activation of the cAMP–PKA pathway. The phosphorylation and activation of adipose triglyceride lipase and hormone-sensitive lipase facilitate lipolysis, resulting in the release of glycerol and free fatty acids into the bloodstream to meet elevated peripheral energy requirements [9].

Circulating asprosin levels are tightly associated with adipose tissue mass and metabolic activity; thus, the hypermetabolic and catabolic condition typical of hyperthyroidism may affect its release. Increased lipolysis and a gradual decrease in adipocyte size may diminish substrate availability and limit the generation of adipocyte-derived hormones, potentially resulting in lowered circulating asprosin levels. Therefore, variations in asprosin levels in hyperthyroid states may indicate complex relationships among excess thyroid hormones, adipose tissue reconfiguration, and overall energy equilibrium [10]. This study aimed to investigate serum asprosin levels in patients with GD and their correlation with thyroid-stimulating hormone receptor antibodies (TRAb), lipid profile parameters, and thyroid hormone levels, including further examination in SCH. Therefore, this study aims to investigate whether serum asprosin can be used as a biomarker to differentiate GD from healthy controls, and to explore its potential role in SCH.

Methods

Study design and subjects

The present case–control study was conducted from July 2025 to January 2026. Sample collection and laboratory analyses were performed at affiliated clinical and academic institutions. Institutional details were omitted to preserve the double-blind peer review process.

Ethical approval

The study protocol was approved by the Research Ethics Committee / Institutional Review Board (IRB) of a university medical institution (Ref. No. 217/3/2; Date: 10 March 2025). Oral informed consent was obtained from all participants prior to enrollment.

Participants

A total of 160 participants aged 22 to 59 years were enrolled in the study. The study included 40 patients with GD and 40 with SCH, determined by a specialist endocrinologist based on clinical assessment and biochemical thyroid function tests. SCH cases included in this study were not classified according to a specific underlying etiology. In addition, 80 apparently healthy euthyroid individuals were included as a control group.

Inclusion criteria

Participants were eligible for inclusion if they matched the following criteria:

- Individuals aged 22 to 59 years.
- GD was diagnosed based on suppressed TSH levels, elevated total thyroxine (T4), and positive TRAb. Only patients presenting with overt hyperthyroidism at the time of evaluation were included. All cases were newly diagnosed and had not received any prior treatment.
- SCH is characterized by suppressed TSH levels with total T4 within the laboratory reference range and clinical signs and symptoms. In the present study, SCH was classified based on TSH and total T4 due to resource limitations and the unavailability of free thyroid hormone measurements. All included patients were newly diagnosed and had not received any prior treatment at the time of enrollment.
- The control group exhibited normal thyroid function testing, with TSH and T4 levels within reference limits, and no known thyroid disease.

Exclusion Criteria

Participants were excluded if they had diabetes mellitus, obesity, or other endocrine disorders; autoimmune diseases excluding thyroid disorders; chronic inflammatory, pulmonary (e.g., COPD), cardiac, renal, or hepatic diseases; malignancy or a history of thyroid cancer; pregnancy; current use of antithyroid, immunosuppressive, anti-inflammatory medications, or mineral supplements; or an inability to provide informed consent.

Sample Size Calculation

The sample size was calculated a priori using G*Power software (version 3.1.9.4; Heinrich Heine University Düsseldorf, Germany) [11]. A one-way ANOVA (fixed effects, omnibus) was conducted, considering a modest effect size ($f = 0.30$) in accordance with Cohen's guidelines [12]. The required minimum sample size, with a significance level of 0.05 and 90% statistical power, was 144 people. To account for any dropouts and absent data, an extra 10% was included, giving a final target sample of 160 participants.

Sample Collection

Following an overnight fast of 10–12 hours, 5 mL of venous blood was collected from each participant in gel separator tubes. Samples were allowed to clot and then centrifuged at 3000 rpm for 10 minutes to obtain serum. Serum aliquots were distributed as follows: 1 mL was used for lipid profile analysis, and 1.5 mL was allocated for thyroid function assessment, including TSH, T4, and TRAb. The remaining serum was aliquoted into sterile Eppendorf tubes and stored at –20°C until further biochemical analysis.

Biochemical and Hormonal Analyses

Serum asprosin concentrations were quantified using a commercially available sandwich enzyme-linked immunosorbent assay (ELISA) kit (MyBioSource, USA), following the manufacturer's instructions. The assay had a detection range of 0.156–10 ng/mL, with a minimum detectable concentration of 0.062 ng/mL. The intra-assay and inter-assay coefficients of variation (CVs) were < 10% and < 12%, respectively. Thyroid function parameters, including TSH and T4, were measured using the VIDAS automated immunoassay system (bioMérieux, France). TRAb was determined using the Roche Cobas e411 analyzer (Roche Diagnostics, Germany). Lipid profile parameters, including total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and very-low-density lipoprotein cholesterol (VLDL-C), were analyzed by enzymatic colorimetric methods using the Roche Cobas c111 automated chemistry analyzer (Roche Diagnostics, Germany). All assays were performed in accordance with standardized laboratory procedures, and internal quality control measures were applied throughout the study period. The reference ranges applied in this study were TSH from 0.27 to 4.20 mIU/L, total T4 from 66 to 181 nmol/L, and TRAb from 0 to 1.75 IU/L, in accordance with the manufacturer's instructions and the laboratory's standard reference values, and for lipid profile parameters TC less than 200 mg/dL, TG less than 150 mg/dL, HDL-C at least 40 mg/dL for men and at least 50 mg/dL for women, LDL-C less than 100 mg/dL, and VLDL-C 5 to 40 mg/dL according to standard laboratory reference values.

Serum asprosin levels were measured using a commercially available ELISA kit according to the manufacturer's instructions. Currently, no universally established reference ranges for circulating asprosin exist, particularly in the context of thyroid disorders. Therefore, in the present study, the control group was used as a comparative reference to interpret relative differences across study groups.

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics (version 25.0; IBM Corp., Armonk, NY, USA). Data distribution normality was assessed using the Shapiro–Wilk test. Continuous variables were expressed as mean ± standard deviation (SD) for normally distributed data. Comparisons among the three study groups (GD, SCH, and healthy controls) were conducted using one-way analysis of variance (ANOVA) for parametric variables, followed by Tukey's post hoc test for multiple comparisons where appropriate. Spearman's rank correlation coefficient (r) was used to evaluate associations between serum asprosin levels and other clinical and biochemical parameters. Receiver operating characteristic (ROC) curve analysis was performed to assess the diagnostic performance of serum asprosin in differentiating patients with GD and SCH from healthy controls. The area under the curve (AUC), optimal cutoff value, sensitivity, and specificity were determined. A two-tailed p-value < 0.05 was considered statistically significant.

Results

As shown in Table 1, there were no statistically significant differences in age, sex, and BMI distribution between the GD and SCH and control groups (P > 0.05).

Table I. – Baseline clinical characteristics of GD, SCH, and control groups

Parameter	GD (n = 40)	SCH (n = 40)	Control (n = 80)	P-value
Sex (M/F)	19/21	18/22	34/46	0.98
Age (years)	39.2 ± 8.1	41.4 ± 8.3	39.8 ± 10.8	0.975
BMI (kg/m^2)	23.9 ± 2.8	24.2 ± 3.1	23.7 ± 2.9	0.68

Serum asprosin levels were significantly lower in patients with GD and SCH compared to controls (p < 0.001 for both). The GD and SCH groups did not differ significantly. Detailed results are presented in Table 2 and illustrated in Figure 1.

Table II. – Comparison of serum asprosin levels among GD, SCH, and control groups

Group	Asprosin (ng/mL)	p-value vs Control
GD	0.56 ± 0.31	< 0.001
SCH	0.65 ± 0.37	< 0.001
Control	2.63 ± 0.52	-

Table 3 illustrates that thyroid function parameters showed significant differences among the study groups (p < 0.001). Patients with GD demonstrated significantly decreased TSH levels and significantly higher total T4 concentrations in comparison to patients with SCH and healthy controls. Moreover, TRAb levels were significantly elevated in the GD group compared to the other groups.

Table III. – The thyroid function parameters and TRAb levels among GD, SCH, and control groups

Parameter	GD	SCH	Control	p-value
TSH (mIU/L)	0.020 ± 0.010	0.149 ± 0.035	2.280 ± 0.733	< 0.001
Total T4 (nmol/L)	204.0 ± 17.2	147.5 ± 10.2	81.5 ± 9.1	< 0.001
TRAb (IU/L)	11.78 ± 5.76	2.33 ± 0.52	0.54 ± 0.33	< 0.001

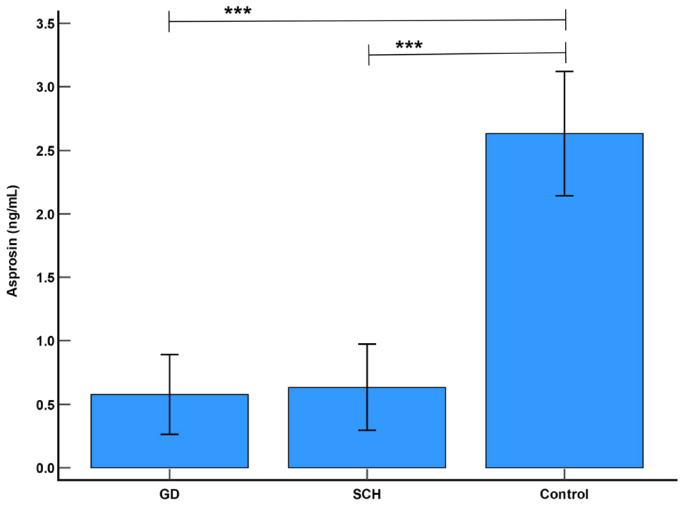

Figure 1. Serum Asprosin Levels in GD, SCH, and Control Groups

Data are presented as mean ± SD. Serum asprosin levels were significantly lower in GD and SCH compared with controls (highly significant, p < 0.001), with no significant difference between GD and SCH.

Table 4 illustrates that lipid profile parameters indicated significant differences among the study groups (p < 0.001 for all comparisons). Patients with GD and SCH demonstrated significantly lower levels of TC, TG, HDL-C, LDL-C, and VLDL-C in comparison to the control group.

Table IV. – Lipid profile parameters among GD, SCH, and control Groups

Parameter	GD	SCH	Control	p-value
TC (mg/dL)	160.6 ± 8.9	166.8 ± 6.7	194.0 ± 17.4	< 0.001
TG (mg/dL)	92.6 ± 11.3	98.4 ± 6.1	114.7 ± 16.7	< 0.001
HDL-C (mg/dL)	38.7 ± 4.0	41.5 ± 3.1	51.8 ± 5.8	< 0.001
LDL-C (mg/dL)	105.3 ± 12.2	108.1 ± 7.7	124.2 ± 8.3	< 0.001
VLDL-C (mg/dL)	13.8 ± 1.0	19.6 ± 1.3	23.0 ± 3.4	< 0.001

Table 5 illustrates that multivariate logistic regression analysis identified serum asprosin, T4, TSH, and TRAb as independent predictors of disease state (p < 0.001 for all). Asprosin and TSH showed inverse correlation with disease risk, but T4 and TRAb demonstrated significant positive correlations.

Table V. – Multivariate logistic regression analysis of factors associated with disease status

Variable (unit)	β(Coefficient)	SE	OR	95% CI for OR	p-value
Asprosin (ng/mL)	–0.534	0.120	0.586	0.46–0.74	<0.001
T4 (nmol/L)	0.557	0.130	1.745	1.35–2.25	<0.001
TSH (mIU/L)	–0.875	0.200	0.417	0.28–0.62	<0.001
TRAb (IU/L)	0.171	0.040	1.186	1.10–1.28	<0.001

Serum asprosin exhibited significant differentiation capacity for GD (Figure 2A), with an AUC of 0.938 (95% CI: 0.824–1.000; p < 0.0001). The cutoff value (< 1.48 ng/mL) produced 87.5% sensitivity and 87.5% specificity. Serum asprosin showed significant distinguishing capacity for detecting SCH (Figure 2B), with an AUC of 0.902 (p < 0.0001). With a cutoff value of < 1.56 ng/mL, the sensitivity and specificity were 90.0% and 81.25%, respectively. Despite this strong performance, these results should be interpreted with caution and require validation in larger, independent cohorts.

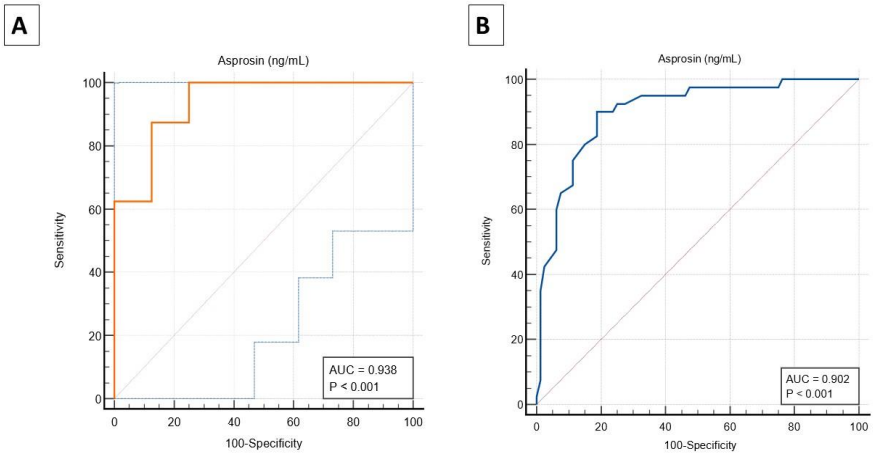

Figure 2. Receiver operating characteristic (ROC) curves of serum asprosin levels.

(A) Differentiation of GD from healthy controls (AUC = 0.938, p < 0.001).

(B) Differentiation of SCH from healthy controls (AUC = 0.902, p < 0.001).

Correlation Analysis

Spearman's rank correlation analysis revealed no statistically significant correlations between serum asprosin levels and thyroid function parameters (TSH, total T4, or TRAb) nor with lipid profile components in patients with GD or SCH ($p > 0.05$ for all comparisons).

Discussions

The present study investigated circulating asprosin levels in patients with GD and SCH relative to healthy controls. The results revealed significant changes in serum asprosin levels across the examined groups, indicating a possible link between thyroid dysfunction and asprosin regulation. The findings suggest that alterations in thyroid hormone levels may affect the secretion or control of metabolic adipokines, including asprosin.

Multivariate analysis further showed that asprosin remained significantly associated with disease status after adjustment for key thyroid-related variables. Although BMI was not included in the regression model, it did not differ significantly between groups, which may have partially mitigated its confounding effect.

To the best of our knowledge, few studies have examined circulating asprosin levels in thyroid dysfunction, and none have directly compared GD, SCH, and healthy controls. Consequently, the present findings provide preliminary clinical evidence of a potential link between thyroid function and asprosin.

Asprosin is an adipokine produced throughout fasting, mostly secreted by white adipose tissue, essential for metabolic homeostasis by enhancing hepatic glucose release and regulating appetite. Thyroid hormones are crucial regulators of energy metabolism, influencing glucose use, lipid metabolism, and basal metabolic rate. Thus, fluctuations in thyroid hormone levels may affect the secretion or regulation of adipokines such asprosin, potentially elucidating the discrepancies in circulating asprosin levels noted in this study.

Hyperthyroidism induces hypermetabolism, elevating the basal metabolic rate, lipolysis, and gluconeogenesis, often resulting in weight loss despite heightened appetite [13]. These metabolic changes may reduce the mass of asprosin-secreting adipose tissue [14]. Moreover, hyperthyroidism-induced increases in sympathetic tone and insulin sensitivity may suppress asprosin synthesis and secretion, thus reducing hepatic glucose production [15, 16]. These mechanisms likely elucidate the diminished circulating asprosin observed in hyperthyroid patients. The findings of this study should be interpreted in the context of GD patients presenting with overt hyperthyroidism, as included in our cohort.

Serum asprosin is not proposed as a replacement for established diagnostic markers such as TSH or TRAb. Rather, it may represent a potential supportive biomarker reflecting metabolic alterations associated with hyperthyroid states. However, its clinical utility remains uncertain and is currently limited to research settings. It may be considered, in future studies, as an adjunct exploratory marker in the evaluation of metabolic aspects of thyroid dysfunction. This study is exploratory in nature and provides preliminary evidence regarding the behavior of asprosin in thyroid dysfunction, without implying clinical applicability at this stage.

Study limitations

This study has several limitations. Firstly, the sample size was limited, and the cross-sectional design prevents causal inferences regarding thyroid function and circulating asprosin. Secondly, potential confounding variables, including nutrition, physical activity, and body composition, were not sufficiently controlled. Significantly, body composition parameters, including fat percentage, were not evaluated, despite asprosin functioning as an adipomyokine and potentially being influenced by metabolic tissue distribution.

Body composition analysis and comprehensive metabolic profiling were not performed due to resource limitations. Instead, BMI was used as a surrogate marker; however, it does not fully reflect adiposity or metabolic status. Importantly, no statistically significant differences in BMI were observed between the study groups, which may partially reduce the potential confounding effect related to differences in adiposity.

Third, only serum asprosin and limited thyroid function parameters were assessed. Free thyroid hormones (fT4 and fT3) were not measured due to laboratory and resource constraints, and only total T4 was available, which may limit the precise assessment of thyroid functional status. In particular, the absence of fT3 may reduce the ability to identify conditions such as T3-toxicosis or subtle thyrotoxic states.

However, the diagnosis of GD and SCH was based on a combination of clinical assessment and TRAb measurements, which strengthens the diagnostic classification of the study groups.

Fourth, only serum asprosin was evaluated, without examining tissue-level expression or downstream metabolic effects, thus limiting mechanistic interpretation. The quantification of asprosin relied on a single commercial ELISA kit without inter-assay calibration against a standardized reference, which may introduce methodological variability. Additionally, no previous studies have evaluated asprosin levels in patients with thyroid dysfunction for direct comparison, further limiting external validation. The absolute values obtained may therefore vary if measured using different assays or in different laboratories, which should be considered when interpreting the results.

Fifth, clinical variables, including disease duration and severity, were not available and therefore could not be assessed. The absence of these data may influence metabolic interpretation and represents a limitation of the present study.

Therefore, due to these limitations, larger longitudinal

studies incorporating comprehensive thyroid profiling (including fT4 and fT3), detailed metabolic and body composition assessments, and standardized validated measurement techniques are necessary to confirm and extend these findings.

Conclusion

This study demonstrates that circulating asprosin levels are decreased in patients with GD and SCH. These findings suggest that serum asprosin may serve as a supportive biomarker for differentiating GD from healthy individuals. Additionally, the observed alterations in asprosin levels may reflect underlying metabolic changes associated with hyperthyroidism. However, further large-scale and longitudinal studies are required to validate its clinical utility and to better understand the relationship between asprosin, thyroid function, and metabolic regulation.

Authors' contributions

S.K.A. (Conceptualization; Data curation; Formal analysis; Investigation; Methodology; Project administration; Resources; Validation; Visualization; Writing – original draft; Writing – review & editing)

M.I.H. (Conceptualization; Formal analysis; Investigation; Methodology; Supervision; Writing – review & editing)

M.S.K. (Investigation; Resources; Writing – review & editing; Supervision)

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Conflict of interest

The authors declare no competing interests.

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