RESEARCH ARTICLE

CYPRUS
JOURNAL OF MEDICAL SCIENCES

DOI: 10.4274/cjms.2026.2025-133
Cyprus J Med Sci 2026;11(2):154-160

Comparative Diagnostic Utility of Systemic Inflammatory Indices Across Hyperthyroidism Subgroups

Müzeyyen Eryılmaz[1], Arzu Güntürk[2], Kadir Yılmaz[3]

[1]Clinic of Internal Medicine, University of Health Sciences Türkiye, Fatih Sultan Mehmet Training and Research Hospital, İstanbul, Türkiye
[2]Clinic of Internal Medicine, Memorial Şişli Hospital, İstanbul, Türkiye
[3]Department of Statistics, İstanbul Commerce University, İstanbul, Türkiye

Abstract

BACKGROUND/AIMS: In this retrospective study, we aimed to examine the diagnostic performance of systemic inflammation response index (SIRI), systemic immune inflammation index (SII), neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) parameters in terms of subgroups in hyperthyroidism cases.

MATERIALS AND METHODS: A total of 191 patient files were included in the study; these patients had applied to the Internal Medicine Department of Fatih Sultan Mehmet Hospital and the Internal Medicine and Endocrinology Department of Florence Nightingale Ataşehir Hospital were diagnosed with hyperthyroidism between January 2023 and June 2023. We evaluated age, gender, chronic drug usage, comorbidity, free T3, T4, anti-thyroid peroxidase (anti-TPO), anti-thyroglobulin antibody (anti-TG), thyrotropin receptor antibodies (TRAb), neutrophil, lymphocyte, platelet (PLT), C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), NLR, PLR, SIRI, and SII parameters of the patients.

RESULTS: Seventy-four patients (89.2%) had hashitoxicosis (HT), 28 patients (33.7%) had Graves' disease (GD), 16 patients (19.3%) had subacute thyroiditis (SAT), and 32 patients (38.6%) had overlapping entries in their files. Only the free T4 level was significantly correlated with hyperthyroidism ($r=0.288$, $p<0.01$). Correlations between hyperthyroidism and age, gender, chronic drug usage, comorbidity, free T3 and free T4, anti-TPO, anti-TG, TRAb, neutrophils, lymphocytes, PLT, CRP, ESR, NLR, PLR, SIRI, and SII were not statistically significant ($p>0.05$). The diagnostic performance of NLR, PLR, SIRI, and SII for hyperthyroidism was not statistically significant ($p>0.05$). Graves was significantly correlated with monocytes ($r=0.240$, $p<0.05$) and with SIRI ($r=0.221$, $p<0.05$). SAT was significantly correlated with CRP ($r=0.574$, $p<0.01$) and ESR ($r=0.626$, $p<0.01$). Overlap was significantly correlated with CRP ($r=0.409$, $p<0.01$) and with ESR ($r=0.246$, $p<0.01$). SIRI had a diagnostic performance of 63.5% for Graves ($p<0.05$). The diagnostic performance of all other blood parameters for HT, GD, SAT, and overlap groups was not significant ($p>0.05$). The area under the curve (AUC) for SIRI in GD was 0.635. At a SIRI cut-off value of 3.70, the sensitivity was 78.6% and the specificity was 52.7%.

CONCLUSION: Although blood parameters lack sufficient diagnostic performance to distinguish hyperthyroidism subtypes, SIRI may have diagnostic performance specific to GD. Despite the low diagnostic performance, it would be beneficial to examine the diagnostic value of SIRI for GD in larger sample sizes and multicenter studies. SIRI should be interpreted as an adjunct biomarker rather than a diagnostic test.

Keywords: Hyperthyroidism, blood parameters, NLR, PLR, SIRI, SII

To cite this article: Eryılmaz M, Güntürk A, Yılmaz K. Comparative diagnostic utility of systemic inflammatory indices across hyperthyroidism subgroups. Cyprus J Med Sci. 2026;11(2):154-160

ORCID IDs of the authors: M.E. 0000-0002-1027-3200; A.G. 0000-0002-1941-8699; K.Y. 0000-0003-2568-3015.

Corresponding author: Müzeyyen Eryılmaz
E-mail: drmuzeyyeneryilmaz@hotmail.com
ORCID ID: orcid.org/0000-0002-1027-3200

Received: 03.11.2025
Accepted: 12.02.2026
Publication Date: 22.04.2026

INTRODUCTION

Hyperthyroidism is a disease characterized by elevated triiodothyronine (T3) and free thyroxine (FT4) concentrations and suppressed thyroid-stimulating hormone (TSH) levels. Low TSH, even with normal T3 and FT4 levels, is defined as subclinical hyperthyroidism and is seen in 0.7% to 1.4% of the population.[1] Graves' disease (GD) is the most common cause of hyperthyroidism; other common causes include drug side effects, toxic multinodular goiter, thyroiditis, and toxic adenomas.[2] Furthermore, iodine intake is the most significant risk factor; smoking, advanced age, ethnicity, genetic predisposition, immune inhibitors, and endocrine disruptors are other risk factors.[3] Hypothalamic and pituitary lesions, ectopic production of thyroid-stimulating proteins by neoplasms outside the thyroid, exogenous drug reactions, and mechanical destructive pathologies are also cited as causes and risk factors in the literature.[4] The clinical presentation often includes arrhythmias (including tachycardia), weight loss, sweating, tremors, anxiety, and palpitations.[5] Common treatments include antithyroid drugs such as propylthiouracil and methimazole, thyroidectomy, or radioactive iodine ablation.[6]

Chronic autoimmune thyroiditis, one of the most common causes of chronic hypothyroidism, has been associated with a transient hyperthyroid phase, termed hashitoxicosis (HT).[7] HT has been reported in 4.2% of cases of unclassified hyperthyroidism.[8] HT has been reported to progress to Hashimoto's thyroiditis.[9] GD, one of the most common causes of hyperthyroidism, has been reported to trigger hyperactivity of the adrenergic nervous system, which is associated with cognitive dysfunction, ophthalmopathy, and goiter.[10] Subacute thyroiditis (SAT), on the other hand, is an inflammatory disease of the thyroid; its clinical course and pathogenesis are still poorly understood.[11] Treatment of hyperthyroidism requires accurate diagnosis and effective management.[12-14] Laboratory and clinical parameters, along with the physician's physical examination and experience, play an important role in diagnosing hyperthyroidism.[15] However, distinctive indicators are still needed in this regard. Although studies have examined the diagnostic and predictive value of hyperthyroidism, studies addressing blood parameters and systemic immune-inflammation indices are lacking. The inflammatory nature of the SAT type and recent, limited studies examining the role of blood parameters in mortality in autoimmune types[16] suggest that these parameters may have diagnostic utility for predicting hyperthyroidism. Therefore, this study aimed to examine the diagnostic performance of the systemic inflammation response index (SIRI), the systemic immune inflammation index (SII), the neutrophil-to-lymphocyte ratio (NLR), and the platelet-to-lymphocyte ratio (PLR) across subgroups of hyperthyroid patients. This study is the first in the literature to evaluate the diagnostic performance of the SIRI, the SII, the NLR, and the PLR across subgroups of hyperthyroidism cases at the multivariate level.

MATERIALS AND METHODS

Research Model

The study had a retrospective, descriptive screening design to identify patients' blood parameters and systemic immune-inflammatory indices. The correlational screening model analyzed the diagnostic performance of blood parameters for hyperthyroidism.

Patients

A total of 191 patient files from the Internal Medicine Department of Fatih Sultan Mehmet Hospital and the Internal Medicine and Endocrinology Departments of Florence Nightingale Ataşehir Hospital were diagnosed with hyperthyroidism between January and June 2023. Based on the study closest to ours by Wang et al.,[16] an effect size of 0.5 (95% confidence interval), a critical t value of 1.6802300 (calculated using G*Power 3.1.9.2), and a minimum sample size of 45 were obtained. This number was significantly exceeded in the study, with 191 patients included.

The inclusion criteria were as follows:

- Over 18 years of age
- Diagnosed with hyperthyroidism
- With complete data in their file
- No non-steroidal anti-inflammatory drugs or steroids have been started
- Not taking steroids or chemotherapy for rheumatological, oncological, or other reasons
- No active infection

Exclusion criteria were as follows:

- Under 18 years of age
- With incomplete data
- Using steroids or anti-inflammatory drugs
- Using steroids or chemotherapy for rheumatological, oncological, or other reasons

TSH levels below 0.4 mU/L are considered indicative of hyperthyroidism. HT was defined as elevated anti-thyroid peroxidase (anti-TPO) (≥5.61 IU/mL) or anti-thyroglobulin antibody (anti-TG) (≥4.11 IU/mL) levels. GD was identified by thyrotropin receptor antibodies (TRAb) ≥1.7 IU/L, and SAT was diagnosed when CRP ≥5 mg/L or erythrocyte sedimentation rate (ESR) ≥20 mm/h. Cases fulfilling more than one criterion were designated as overlapping phenotypes, including the HT+GH, HT+SA, and HT+GH+SA subgroups.

NLR = Neutrophil count/lymphocyte count ratio;

PLR = Platelet (PLT) count/lymphocyte count ratio;

SII = (PLT count × neutrophil count)/lymphocyte count ratio.

The following parameters of the patients were evaluated: age, gender, chronic drug use, comorbidities, free T3 and free T4, anti-TPO, anti-TG, TRAb, neutrophil and lymphocyte counts, PLT, CRP, ESR, NLR, PLR, SIRI, and SII.

The research algorithm initially involved defining and analyzing the dataset. Subsequently, a correlational screening analysis was performed on biomarker values of blood parameters and their ratios.

In examining the relationships between patients' blood parameters, ratios and logit-model analysis were used to minimize deflator effects arising from different units. This aimed to minimize unit conversion errors and systematic errors.

Ethical Approval

Ethical approval was obtained from Demiroğlu Science University Clinical Research Ethical Committee (approval number: 44140529/34423, date: 05.12.2023). Patient consent was not obtained because the study was

retrospective. The Declaration of Helsinki and good clinical practice procedures were followed in the study.

Statistical Analysis

Patients' nominal and ordinal parameters were described using frequencies, while measurement parameters were described using means and standard deviations. The Kolmogorov-Smirnov test was used to assess whether the measurement parameters conformed to a standard normal distribution. An Independent Samples t-test was used to compare parameters with a normal distribution, and the Mann-Whitney U test was used to compare parameters that did not conform to a normal distribution. Fisher's exact test was used to analyze differences between nominal and ordinal variables. Since the hyperthyroidism (yes/no) and Graves (yes/no) parameters are dummy variables, Spearman's rho correlation analysis was used. Due to linearization deviations,[17,18] Spearman's rho correlation analysis was used in the relational screening analysis. Receiver operating characteristic (ROC) analysis was performed to assess diagnostic performance. All analyses were performed using SPSS 25.0 for Windows with 95% confidence intervals and a significance level of 0.05. Since our study relies on relationship analysis and does not include between-group comparisons, we did not use Bonferroni or FDR corrections.

RESULTS

74 patients (89.2%) had HT, 28 (33.7%) had GD, 16 (19.3%) had SAT, and 32 (38.6%) had overlap, among patients with values recorded in their files. The mean age of patients without hyperthyroidism was 59.67±13.49 years, and the mean age of patients with hyperthyroidism was 56.78±13.77 years. Females comprised 93.3% of patients without hyperthyroidism and 83.8% of patients with hyperthyroidism. Differences in age, gender, chronic drug usage, comorbidity, free T3 and T4, anti-TPO, anti-TG, TRAb, neutrophil, lymphocyte, PLT, CRP, ESR, NLR, PLR, SIRI, and SII between hyperthyroidism subtypes were not significant ($p>0.05$) (Table 1).

Spearman's rho correlation analysis showed that only the Free T4 level was significantly correlated with hyperthyroidism ($r=0.288$, $p<0.01$). No statistically significant correlations were found between hyperthyroidism and age, gender, chronic drug usage, comorbidity, free T3, free T4, anti-TPO, anti-TG, TRAb, neutrophil, lymphocyte, PLT, CRP, ESR, NLR, PLR, SIRI, and SII ($p>0.05$; Table 2).

Spearman's rho correlation analysis showed that Graves' correlated significantly with monocytes ($r=0.240$, $p<0.05$) and SIRI ($r=0.221$, $p<0.05$). SAT was significantly correlated with CRP ($r=0.574$; $p<0.01$) and ESR ($r=0.626$; $p<0.01$). Overlapping was significantly correlated with CRP ($r=0.409$, $p<0.01$) and with ESR ($r=0.246$, $p<0.01$) (Table 3).

ROC analysis showed that SIRI had a diagnostic performance of 63.5% for GD ($p<0.05$). Diagnostic performance of all other blood parameters in HT, GD, SAT, and overlap groups was not significant ($p>0.05$) (Table 4).

The mean SIRI in the Graves group was higher, while the range of SIRI in Graves' patients was lower than in patients without Graves' (Figure 1).

Area under the curve (AUC) was 0.635 for SIRI for Graves. For a SIRI cut-off level of 3.70, sensitivity was 78.6% and specificity was 52.7% (Figure 2).

DISCUSSION

This study examined the diagnostic performance of the SIRI, SII, NLR, and PLR across subgroups of patients with hyperthyroidism. The files of 191 patients were analyzed. The results showed that the SIRI parameter demonstrated statistically significant diagnostic performance for GD.

Hyperthyroidism is a disease affecting between 0.2% and 1.4% of the general population worldwide1, with risk factors ranging from thyroid lesions and other thyroid diseases to general risk factors and demographics.[2,3] Because the clinical presentation often includes features seen in common diseases, such as arrhythmia, weight loss, sweating, and anxiety,[5] additional biomarkers or diagnostic criteria are needed to facilitate differential diagnosis.

HT occurs in approximately 4.2% of unclassified hyperthyroidism cases;[7] GD is among the most common causes;[8] SAT is a lesser-known inflammatory disease.[11] The diverse characteristics of these subtypes and etiologies, along with the differences in treatment approaches, highlight the importance of differential diagnosis and diagnostic performance in achieving successful treatment and preventing disease progression. However, studies on this topic are limited, and more biomarkers and improved diagnostic performance are needed for clinical applications. Although studies on this topic are limited, they highlight the importance of these findings. Among these, Wang et al.[16] reported that SIRI and SII parameters are statistically significant and useful biomarkers in patients with autoimmune thyroiditis. In our study, the diagnostic performance of SIRI for GD was also significant. In another study, Wang et al.[19] reported that the SIRI and SII parameters have significant diagnostic performance for thyroid cancer. He et al.[20] examined the predictive value of blood parameters for SAT and reported that the NLR, PLR, MLR, SII, and SIRI indices were higher in the SAT group. In this study, however, differences in ESR and thyroid peroxidase antibody were not significant among SAT patients. In this respect, the findings are consistent with our study.

Zhai et al.[21] examined the relationship between thyroid function and SIRI and reported that inflammation may trigger the development and progression of thyroid disorders, suggesting potential diagnostic utility. However, diagnostic performance has not been directly studied; the relationship between inflammation and thyroid progression has been demonstrated by correlation. Piticchio et al.[22] analyzed the inflammatory profile in an athyreotic population with a history of hypothyroidism and reported that the median values of blood inflammatory markers may be predictive of thyroid cancer, particularly among obese individuals. Munteanu et al.[23] examined the role of indices derived from the CBC parameters in the evaluation of Hashimoto's thyroiditis in children. In their study, the NLR was reported as a diagnostic index for Hashimoto's thyroiditis in pediatric patients. Soyer et al.[24] examined the predictive value of blood parameters for SAT and GD and reported that these parameters had significant power to discriminate between the two diseases. Our study also yielded similar results, with SIRI demonstrating significant diagnostic performance for GD. Although recent studies have examined the diagnostic performance of blood parameters in hyperthyroidism, few have comprehensively evaluated their ability to discriminate among subtypes. Therefore, our results suggest that SIRI may have significant diagnostic performance for GD.

Study Limitations

The most significant limitation of the study is the limited literature on the diagnostic performance for hyperthyroidism, which reduces the number of studies available for comparison with our results. Although this places the study among pioneering work, further multicenter studies are needed in this area with larger sample sizes.

Another significant limitation of the study is its retrospective nature. Therefore, the parameters used in the study were based on data compiled from patient files that represented the majority of patients. However, our results suggest that further prospective studies, particularly in patients with GD, would be beneficial. The relatively small subgroup sizes may have limited the detection of additional predictive markers. Although

Table 1. Baseline characteristics and clinical parameters of patient groups with difference analysis results

	Hyperthyroidism		p-value
	No (n=15; 18.1%)	Yes (n=68; 81.9%)	
Age, years, mean ± SD Median (min-max)	59.67±13.49 59.00 (41.00-79.00)	56.78±13.77 59.00 (21.00-81.00)	0.463[a]
Gender, n (%)			
Female	14 (93.3)	57 (83.8)	0.313[b]
Male	1 (6.7)	11 (16.2)	
Chronic drug usage, n (%)	11 (73.3)	55 (80.9)	0.366[b]
Comorbidity, n (%)	7 (46.7)	32 (47.1)	0.603[b]
TSH, mUI/mL	1.33±0.94 0.95 (0.42-3.25)	0.12±0.10 0.11 (0.01-0.38)	0.000[c]
Free T3, ng/dL	2.91±0.42 2.94 (1.96-3.58)	3.44±1.10 3.11 (2.05-6.83)	0.189[c]
Free T4, ng/dL	1.22±0.28 1.18 (0.57-1.94)	1.61±0.66 1.48 (0.53-4.79)	0.009[c]
Anti-TPO, IU/mL	32.47±40.14 16.00 (0.68-139.00)	90.26±193.25 12.40 (0.48-986.00)	0.896[c]
Anti-TG, IU/mL	48.34±63.42 20.00 (0.90-190.00)	100.03±261.55 15.50 (0.90-1731.00)	0.896[c]
TRAb, IU/L	5.92±10.62 0.80 (0.28-31.53)	4.10±6.25 1.07 (0.10-22.80)	0.703[c]
Neutrophil, µL	4.21±0.88 4.30 (3.00-6.30)	4.35±1.47 3.90 (1.70-10.40)	0.999[c]
Lymphocyte, µL	2.27±0.66 2.10 (1.50-4.10)	2.33±0.67 2.30 (0.70-4.40)	0.420[c]
Monocyte, µL	0.47±0.12 0.50 (0.30-0.80)	0.47±0.14 0.49 (0.20-0.80)	0.885[c]
PLT, µL	255.33±55.09 252.00 (175.00-359.00)	276.57±60.49 265.00 (113.00-456.00)	0.227[c]
CRP, mg/L	3.23±3.25 1.61 (0.60-12.00)	4.83±14.73 1.96 (0.09-116.34)	0.657[c]
ESR, mm/hour	14.33±10.85 13.00 (2.00-37.00)	13.97±8.37 13.50 (2.00-44.00)	0.896[c]
NLR	1.94±0.53 1.74 (1.41-3.07)	2.07±1.06 1.80 (0.69-5.60)	0.749[c]
PLR	118.46±33.20 125.91 (66.55-170.95)	128.94±50.35 122.80 (44.32-380.00)	0.628[c]
SIRI	5.09±4.58 3.28 (1.89-20.66)	5.02±3.52 4.20 (0.86-20.80)	0.767[c]
SII	489.75±147.06 504.06 (303.95-775.10)	576.39±341.75 471.42 (163.85-1976.00)	0.705[c]

[a]Independent Samples t-test, [b]Fisher's exact test, [c]Mann-Whitney U test.
SD: Standard deviation, TSH: Thyroid stimulating hormone, Anti-TPO: Anti-thyroid peroxidase, Anti-TG: Anti-thyroglobulin antibody, TRAb: Thyrotropin receptor antibodies, PLT: Platelet, CRP: C-reactive protein, ESR: Erythrocyte sedimentation rate, NLR: Neutrophil-to-lymphocyte ratio, PLR: Platelet-to-lymphocyte ratio, SIRI: Systemic inflammation response index, SII: Systemic inflammation index.

the power analysis performed on our sample of patients indicates that the sample size is sufficient, a larger sample is needed to improve the generalizability of the results. However, we were able to achieve this number at a single center within this time period. Therefore, it would be beneficial to conduct the study in a multicenter setting with a larger sample size. SAT is defined solely by CRP or ESR levels. Clinical findings (neck pain, fever), ultrasound, and RAI uptake criteria are not included. This situation may create misclassification bias. This deficiency may affect the results and must be evaluated in further research.

Table 2. Spearman's rho correlation analysis results between hyperthyroidism and baseline and clinical parameters

Hyperthyroidism	r	p
Age	-0.069	0.533
Gender	0.104	0.349
Drug usage	0.072	0.518
Comorbidity	0.003	0.978
Free T3	0.145	0.191
Free T4	0.288**	0.008
Anti-TPO	0.014	0.897
Anti-TG	-0.014	0.897
TRAb	0.043	0.706
Neutrophil	0.000	1.000
Lymphocyte	0.089	0.424
Monocyte	-0.016	0.886
PLT	0.133	0.229
CRP	-0.049	0.660
ESR	0.014	0.897
NLR	-0.035	0.751
PLR	0.054	0.630
SIRI	0.033	0.769
SII	0.042	0.707

**p<0.01.
TSH: Thyroid stimulating hormone, Anti-TPO: Anti-thyroid peroxidase, Anti-TG: Antithyroglobulin antibody, TRAb: Thyrotropin receptor antibodies, PLT: Platelet, CRP: C-reactive protein, ESR: Erythrocyte sedimentation rate, NLR: Neutrophil-to-lymphocyte ratio, PLR: Platelet-to-lymphocyte ratio, SIRI: Systemic inflammation response index, SII: Systemic inflammation index.

The Study's Contribution to the Literature and Clinical Practice

The study's most significant contribution to the literature is demonstrating that blood parameters may have discriminatory diagnostic performance across hyperthyroidism subgroups, highlighting the need for further research in this area. The study results also demonstrate the potential diagnostic value of the SIRI parameter for GD.

This study demonstrates the clinical benefit of monitoring the SIRI parameter in patients with GD. Although the AUC values obtained from ROC analysis of SIRI in GD are modest, they are statistically significant. Therefore, while higher AUC values are needed to allow generalization, the results demonstrate the clinical benefit of monitoring SIRI values at the extremes in GD.

CONCLUSION

Although blood parameters lack sufficient diagnostic performance to distinguish hyperthyroidism subtypes, SIRI may have specific diagnostic performance for GD. Although SIRI has low diagnostic performance, larger sample sizes and multicenter studies would be beneficial for examining the predictive and diagnostic value of SIRI for GD. This would allow expanded control and scope of research with more cofounders and could contribute to both the literature and clinical practice of thyroid disorders, including hyperthyroidism. SIRI should be interpreted as an adjunct biomarker rather than a diagnostic test. SIRI showed a statistically significant but clinically modest association with GD.

Table 3. Spearman's rho correlation analysis results for relationship between hyperthyroidism sub groups and blood parameters

	Hashitoxicosis (n=74; 89.2%)		Graves (n=28; 33.7%)		Subacute thyroiditis (n=16; 19.3%)		Overlap (n=32; 38.6%)	
	r	p	r	p	r	p	r	p
Neutrophil	-0.001	0.994	0.078	0.485	0.154	0.165	0.101	0.362
Lymphocyte	0.002	0.988	0.171	0.122	-0.126	0.257	0.045	0.690
Monocyte	-0.156	0.158	**0.240***	0.029	-0.007	0.953	0.067	0.550
PLT	0.034	0.760	-0.104	0.351	-0.004	0.968	-0.035	0.756
CRP	-0.039	0.727	0.143	0.199	**0.574****	0.000	**0.409****	0.000
ESR	-0.087	0.436	-0.084	0.452	**0.626****	0.000	**0.246***	0.025
NLR	-0.024	0.828	-0.068	0.544	0.183	0.098	0.021	0.853
PLR	-0.102	0.359	-0.158	0.154	0.166	0.134	-0.026	0.813
SIRI	-0.063	0.571	**0.221***	0.045	-0.022	0.846	0.075	0.501
SII	-0.036	0.749	-0.035	0.756	0.194	0.079	0.078	0.483

*p<0.05, **p<0.01.
PLT: Platelet, CRP: C-reactive protein, ESR: Erythrocyte sedimentation rate, NLR: Neutrophil-to-lymphocyte ratio, PLR: Platelet-to-lymphocyte ratio, SIRI: Systemic inflammation response index, SII: Systemic inflammation index.

Table 4. ROC analysis results for diagnostic performance of blood parameters on hyperthyroidism subtypes

Test result variable (s)	Area	Std. error	p	Asymptotic 95% confidence interval	
				Lower bound	Upper bound
Hashitoxicosis					
NLR	0.477	0.102	0.826	0.277	0.678
PLR	0.405	0.118	0.356	0.174	0.637
SIRI	0.441	0.109	0.568	0.228	0.655
SII	0.467	0.099	0.747	0.273	0.661
Graves					
NLR	0.459	0.068	0.541	0.325	0.593
PLR	0.404	0.066	0.153	0.274	0.533
SIRI	**0.635**	**0.065**	**0.046**	**0.508**	**0.762**
SII	0.479	0.067	0.754	0.347	0.610
Subacute thyroiditis					
NLR	0.634	0.083	0.098	0.470	0.797
PLR	0.621	0.087	0.133	0.450	0.792
SIRI	0.484	0.089	0.844	0.310	0.658
SII	0.642	0.076	0.079	0.492	0.791
Overlap					
NLR	0.512	0.068	0.852	0.380	0.645
PLR	0.484	0.066	0.811	0.355	0.613
SIRI	0.544	0.066	0.498	0.416	0.673
SII	0.546	0.065	0.480	0.419	0.674

NLR: Neutrophil-to-lymphocyte ratio, PLR: Platelet-to-lymphocyte ratio, SIRI: Systemic inflammation response index, SII: Systemic inflammation index, ROC: Receiver operating characteristic.

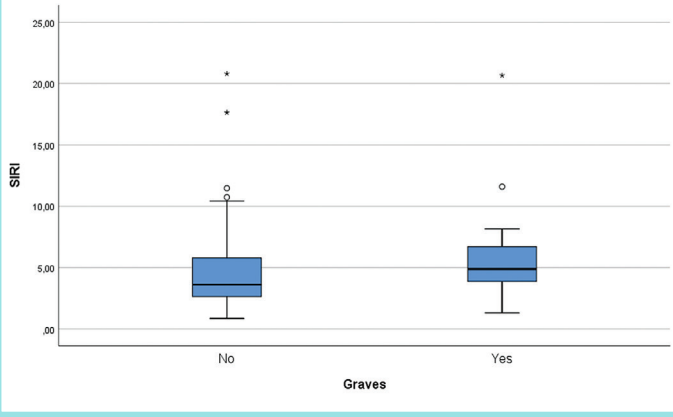

Figure 1. SIRI means and changes according to Graves' groups.
SIRI: Systemic inflammation response index.

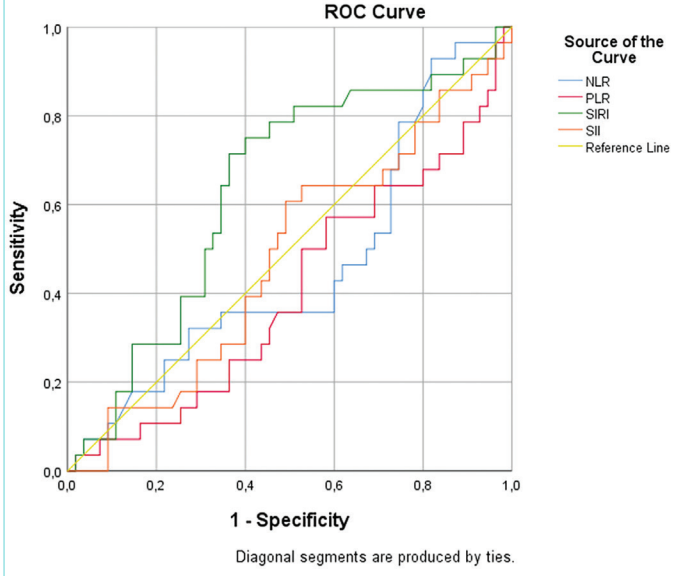

Figure 2. ROC analysis results for NLR, PLR, SIRI and SII.
ROC: Receiver operating characteristic, NLR: Neutrophil-to-lymphocyte ratio, PLR: Platelet-to-lymphocyte ratio, SIRI: Systemic inflammation response index, SII: Systemic inflammation index.

MAIN POINTS

- Hyperthyroidism is a disease affecting between 0.2% and 1.4% of the general population worldwide.
- Biomarkers or diagnostic criteria are needed for a differential diagnosis.
- Blood parameters may have discriminatory diagnostic performance across subgroups of hyperthyroidism.
- Systemic inflammation response index may have specific diagnostic performance for Graves' disease.

ETHICS

Ethics Committee Approval: Ethical approval was obtained from Demiroğlu Science University Clinical Research Ethical Committee (approval number: 44140529/34423, date: 05.12.2023).

Informed Consent: Patient consent was not obtained because the study was retrospective.

Footnotes

Authorship Contributions

Surgical and Medical Practices: M.E., A.G., Concept: M.E., A.G., K.D., Design: M.E., Data Collection and/or Processing: M.E., A.G., Analysis and/or Interpretation: K.D., Literature Search: K.D., Writing: M.E., A.G., K.D.

DISCLOSURES

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

REFERENCES

1. Lee SY, Pearce EN. Hyperthyroidism: a review. JAMA. 2023; 330(15): 1472-83.
2. Reid JR, Wheeler SF. Hyperthyroidism: diagnosis and treatment. Am Fam Physician. 2005; 72(4): 623-30.
3. 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.
4. LiVolsi VA, Baloch ZW. The Pathology of hyperthyroidism. Front Endocrinol (Lausanne). 2018; 9: 737.
5. Guerri G, Bressan S, Sartori M, Costantini A, Benedetti S, Agostini F, et al. Hypothyroidism and hyperthyroidism. Acta Biomed. 2019; 90(10-S): 83-6.
6. Kravets I. Hyperthyroidism: diagnosis and treatment. Am Fam Physician. 2016; 93(5): 363-70.
7. Unnikrishnan AG. Hashitoxicosis: a clinical perspective. Thyroid Research and Practice. 2013; 10(Suppl 1): S5-6.
8. Reinwein D, Benker G, König MP, Pinchera A, Schatz H, Schleusener A. The different types of hyperthyroidism in Europe. Results of a prospective survey of 924 patients. J Endocrinol Invest. 1988; 11(3): 193-200.
9. Shahbaz A, Aziz K, Umair M, Sachmechi I. Prolonged duration of hashitoxicosis in a patient with hashimoto's thyroiditis: a case report and review of literature. Cureus. 2018; 10(6): e2804.
10. Abraham P, Avenell A, Park CM, Watson WA, Bevan JS. A systematic review of drug therapy for Graves' hyperthyroidism. Eur J Endocrinol. 2005; 153(4): 489-98.
11. Stasiak M, Lewiński A. New aspects in the pathogenesis and management of subacute thyroiditis. Rev Endocr Metab Disord. 2021; 22(4): 1027-39.
12. Carlé A, Pedersen IB, Knudsen N, Perrild H, Ovesen L, Rasmussen LB, et al. Epidemiology of subtypes of hyperthyroidism in Denmark: a population-based study. Eur J Endocrinol. 2011; 164(5): 801-9.
13. Wiersinga WM, Poppe KG, Effraimidis G. Hyperthyroidism: aetiology, pathogenesis, diagnosis, management, complications, and prognosis. Lancet Diabetes Endocrinol. 2023; 11(4): 282-98.
14. Bereda G. Hyperthyroidism: definition, causes, pathophysiology and management. Austin J Endocrinol Diabetes. 2022; 9(1): 1091.
15. Mounsey A, Paulus R, Roten W. Hyperthyroidism: diagnosis and treatment. Am Fam Physician. 2025; 112(2): 146-52.
16. Wang B, Wu Z, Hu T. Systemic inflammatory markers predict mortality in autoimmune thyroiditis: threshold-driven risk stratification and prognostic insights from a nationwide cohort. Hormones. 2026; 25: 225-36.
17. Yılmaz K, Turanlı M. A multi-disciplinary investigation of linearization deviations in different regression models. Asian Journal of Probability and Statistics. 2023; 22(3): 15-9.
18. Yilmaz K, Turanlı M. A multi-disciplinary investigation on minimizing linearization deviations in different regression models. Change & Shaping the Future, IV. ASC-2022/Fall Congress
19. Wang Y, Chang J, Hu B, Yang S. Systemic immune-inflammation index and systemic inflammation response index predict the response to radioiodine therapy for differentiated thyroid cancer. J Inflamm Res. 2024; 17: 8531-41.
20. He P, Yang H, Lai Q, Kuang Y, Huang Z, Liang X, et al. The diagnostic value of blood cell-derived indexes in subacute thyroiditis patients with thyrotoxicosis: a retrospective study. Ann Transl Med. 2022; 10(6): 322.
21. Zhai Y, Wang B, Han W, Yu B, Ci J, An F. Correlation between systemic inflammatory response index and thyroid function: 2009-2012 NHANES results. Front Endocrinol (Lausanne). 2024; 14: 1305386.
22. Piticchio T, Savarino F, Volpe S, Prinzi A, Costanzo G, Gamarra E, et al. Inflammatory profile assessment in a highly selected athyreotic population undergoing controlled and standardized hypothyroidism. Biomedicines. 2024; 12(1): 239.
23. Munteanu AI, Jugănaru I, Nicoară DM, Mang N, Vasilescu R, Brad GF, et al. Evaluating the role of cbc-derived indices in children with Hashimoto's thyroiditis. Diagnostics (Basel). 2024; 14(24): 2834.
24. Soyer AK, Cuhaci Seyrek FN, Durmus Demirel K, Tam AA, Topaloglu O, Ersoy R, et al. The role of blood cell derived parameters in the differential diagnosis of subacute thyroiditis and Graves' disease and long-term outcomes in subacute thyroiditis: a tertiary center experience. Endocrine Abstracts. 2025.