Neutrophil-to-Lymphocyte and Platelet-to-Lymphocyte Ratios in Hashimoto Thyroiditis: Biological Rationale, Clinical Evidence, and Interpretive Limits

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Abstract

Background: Hashimoto thyroiditis is sustained by thyroid-directed adaptive immunity, yet its biological effects may extend beyond the gland. The neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) are inexpensive blood count-derived indices proposed to reflect this systemic component.

Objective: This narrative review examines the immunological basis, clinical evidence, limitations, and potential applications of NLR and PLR in Hashimoto thyroiditis.

Synthesis: Neutrophils can amplify tissue injury through inflammatory mediators, whereas lymphocyte redistribution may reflect adaptive immune activation and physiological stress. Platelets contribute to immune-cell recruitment and cytokine signaling, providing a rationale for PLR. Adult studies, however, are inconsistent. Some cohorts found higher NLR and PLR in euthyroid or hypothyroid Hashimoto thyroiditis, whereas others reported unchanged indices or a paradoxically lower PLR. Recent studies also show that thyroid functional state does not reliably stratify these ratios. Differences in obesity, infection exclusion, medication exposure, anemia, nutritional status, smoking, metabolic disease, analytical methods, and patient selection probably contribute to this heterogeneity. Associations with thyroid-stimulating hormone, free thyroxine, thyroid antibodies, C-reactive protein, or erythrocyte sedimentation rate are similarly variable. Neither ratio has a validated universal threshold or sufficient specificity to diagnose Hashimoto thyroiditis, distinguish it from other thyroid disorders, or replace thyroid function tests, antibodies, and ultrasonography.

Conclusion: NLR and PLR are biologically plausible, cost-neutral adjuncts that may describe systemic inflammatory context, but current evidence does not support their independent diagnostic or monitoring use. Prospective, phenotype-specific studies with standardized sampling and multivariable validation are required.

Keywords: Hashimoto thyroiditis; neutrophil-to-lymphocyte ratio; platelet-to-lymphocyte ratio; systemic inflammation; thyroid autoimmunity

Introduction

Hashimoto thyroiditis (HT) is the most prevalent autoimmune thyroid disorder and a leading cause of acquired hypothyroidism in iodine-sufficient populations. A meta-analysis of adult studies documented marked variation in prevalence according to geographic region, age, sex, iodine exposure, diagnostic definition, and study period. The disease is substantially more common in women and may remain biochemically euthyroid for years before progressing to subclinical or overt hypothyroidism. [1,2]

Diagnosis is anchored in thyroid function testing, particularly thyroid-stimulating hormone (TSH) and free thyroxine (FT4), supported by thyroid peroxidase antibodies (TPOAb), thyroglobulin antibodies (TgAb), and characteristic ultrasonographic changes. These measures identify autoimmunity and functional impairment but do not directly quantify the systemic inflammatory milieu. Persistent fatigue, metabolic abnormalities, hematological changes, and cardiovascular risk signals in some patients have therefore prompted interest in inexpensive biomarkers that might complement conventional assessment. [3,4]

The neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) are calculated from routine complete blood counts and integrate cell populations involved in innate immunity, adaptive immunity, thrombosis, and physiological stress. Their accessibility is attractive, particularly in resource-limited practice. The central question, however, is not whether these ratios can change in HT, but whether the changes are sufficiently consistent, disease-specific, and clinically informative to improve diagnosis, phenotyping, or follow-up. [5,6]

Immunopathological Context of Hashimoto Thyroiditis

HT develops through loss of tolerance to thyroid antigens in a genetically susceptible host exposed to modifying environmental factors. Antigen-presenting cells activate autoreactive CD4-positive T cells, while CD8-positive cytotoxic lymphocytes, B cells, macrophages, cytokines, and death-receptor pathways contribute to progressive thyrocyte injury. TPOAb and TgAb are clinically useful signatures of this immune response, although their concentrations do not map directly onto the amount of surviving thyroid tissue or the degree of hormonal failure. [3,4]

Thyroid inflammation is predominantly tissue-centered. Lymphocytic infiltration, germinal-center formation, follicular destruction, Hürthle-cell change, and variable fibrosis can be extensive even when circulating thyroid hormone concentrations remain normal. Conversely, systemic inflammatory markers may be modest because the gland is small and the autoimmune process is chronic rather than an acute neutrophil-dominant reaction. This biological separation between local immune activity and circulating cell counts is fundamental when interpreting NLR and PLR. [4,5]

Evidence nevertheless supports some systemic immune signaling. Circulating proinflammatory cytokines, altered immune-cell phenotypes, oxidative stress, endothelial dysfunction, and associations between antibody concentrations and symptom burden have been reported. In one recent cohort, higher thyroid antibody concentrations were associated with multiple inflammatory and symptomatic measures, but the cross-sectional design could not establish whether antibodies caused the systemic findings or simply marked a broader autoimmune phenotype. [7,8]

Biological Basis of NLR

NLR is calculated by dividing the absolute neutrophil count by the absolute lymphocyte count. Its value can rise through neutrophilia, lymphopenia, or both. Neutrophils respond rapidly to interleukin signaling, stress hormones, infection, tissue injury, smoking, and metabolic inflammation. Lymphocyte counts are influenced by adaptive immune activation, redistribution, apoptosis, corticosteroids, nutritional state, and acute physiological stress. NLR therefore compresses two biologically different processes into one number, improving convenience but reducing mechanistic specificity. [9]

In HT, several pathways could increase NLR. Cytokine-mediated marrow release and delayed neutrophil apoptosis may increase circulating neutrophils, while lymphocyte trafficking from blood into thyroid tissue could reduce the peripheral lymphocyte denominator. Hypothyroidism can add metabolic stress,

dyslipidemia, endothelial dysfunction, and altered hematopoiesis. Yet HT is driven mainly by lymphocytes rather than circulating neutrophils; active lymphocyte production or preserved peripheral counts could therefore blunt or even reverse the expected ratio change. [3,10]

NLR also varies with age, sex, body mass index, time of sampling, exercise, smoking, acute infection, diabetes, renal or hepatic dysfunction, malignancy, hematological disease, and medication exposure. Small differences between HT and control groups can disappear after these factors are balanced or adjusted. A statistically higher mean NLR in a retrospective cohort consequently does not establish a diagnostic biomarker unless discrimination, calibration, reproducibility, and incremental value beyond established thyroid tests are demonstrated. [6,9]

Biological Basis of PLR

PLR is the platelet count divided by the absolute lymphocyte count. Platelets are immune-active cells rather than passive hemostatic fragments. They release chemokines and growth factors, interact with neutrophils and lymphocytes, facilitate leukocyte recruitment, and participate in endothelial inflammation. Interleukin-6 and other mediators may stimulate thrombopoiesis, while lymphocyte redistribution can further raise the ratio. These mechanisms make PLR a plausible signal of combined inflammatory and thromboimmune activity. [11]

Interpretation is complicated because platelet counts are affected by iron deficiency, anemia, menstrual blood loss, inflammation, liver disease, splenic sequestration, autoimmunity, medication, and laboratory variation. Hypothyroidism is associated with normocytic, microcytic, or macrocytic anemia depending on erythropoietic drive, iron and vitamin status, bleeding, and associated autoimmune gastritis. Reactive thrombocytosis from iron deficiency can elevate PLR without indicating more severe thyroid autoimmunity, whereas thrombocytopenia or relatively preserved lymphocyte counts can lower it. [10,11]

Clinical Evidence for NLR and PLR

Early studies generally supported higher ratios. Bilge et al. compared 145 women with euthyroid HT and 60 age-matched controls. NLR was 2.29 ± 0.65 versus 1.68 ± 0.40, and PLR was 164.95 ± 55.14 versus 106.88 ± 32.19; both differences were significant at $P<0.001$. Platelet counts were higher and lymphocyte counts lower in the HT group, showing that the PLR signal was produced by changes in both numerator and denominator. Levothyroxine exposure did not remove the between-group differences. [12]

Aktas et al. studied 90 patients with HT and 64 controls and reported a small but statistically significant NLR difference: median 2.1 (range, 1.3–5.8) versus 1.9 (0.6–3.3), $P=0.04$. The distributions overlapped extensively, illustrating why significance testing alone is insufficient for individual classification. Keskin et al. also reported increased NLR in euthyroid chronic autoimmune thyroiditis, supporting the possibility that circulating changes can precede overt thyroid failure. [13,14]

Findings in hypothyroid HT are not uniform. In a retrospective study of 121 patients with overt or subclinical hypothyroidism and 100 controls, Önalan and Dönder found higher NLR in patients (2.43 ± 0.94 vs 2.11 ± 0.81) but lower PLR (130.8 ± 50.5 vs 145.3 ± 58.5). Neither ratio correlated significantly with TSH, FT4, or TPOAb. The opposite directions of NLR and PLR argue against interpreting both as interchangeable measures of one inflammatory burden. [15]

Later studies strengthened the uncertainty. Resber et al. examined 93 euthyroid patients and 86 controls in an obesity-clinic population. NLR and PLR were numerically higher in HT but not statistically different ($P=0.427$ and $P=0.089$). Treated and untreated patients also had similar NLR and PLR. C-reactive protein (CRP) showed weak positive associations with TPOAb, neutrophils, platelets, and NLR, suggesting that conventional inflammatory activity and CBC ratios may overlap only partially. [16]

PLR-specific data are equally divergent. Erge et al. reported median PLR values of 177 (range, 72–417) in hypothyroid-thyrotoxic HT, 137 (69–272) in euthyroid HT, and 103 (44–243) in controls ($P<0.001$), with a strong positive association between PLR and CRP. In contrast, the hypothyroid cohort of Önalan and Dönder and the larger analysis by Bozdag and Gundogan Bozdag found lower PLR in selected HT groups than in controls. [15,17]

A broader comparison by Erinc et al. included 166 euthyroid HT patients, 91 hypothyroid HT patients,

58 patients with nonimmune hypothyroidism, and 190 controls. Systemic immune-inflammation index and PLR differed from controls, but NLR did not reach overall significance (P=0.059). None of these indices reliably separated euthyroid HT, hypothyroid HT, and nonimmune hypothyroidism, suggesting that thyroid hormone deficiency and comorbidity can generate signals similar to thyroid autoimmunity. [18]

Bozdag and Gundogan Bozdag analyzed 268 patients with HT—131 euthyroid, 83 hypothyroid, and 54 hyperthyroid—plus 124 controls. Median NLR was higher in every HT subgroup than in controls: 1.95, 2.17, and 1.95 versus 1.60, respectively (overall P<0.001). PLR moved in the opposite direction, with medians of 136.19, 124.06, and 113.32 compared with 144.90 in controls (overall P=0.002). None of the studied inflammatory indices distinguished the thyroid functional subgroups. [19]

The most recent adult evidence remains cautious. Demir and Taştemur compared 92 patients with HT and 92 controls and found no significant differences in NLR, PLR, or systemic immune-inflammation index. ESR, monocyte count, hemoglobin, and lymphocyte-to-monocyte ratio (LMR) differed, while LMR and the FT4/TSH ratio emerged as independent predictors. Anti-TPO and anti-Tg had excellent apparent discrimination, but the authors appropriately noted incorporation bias because antibody positivity contributed to disease classification. [20]

Age may modify the signal. In a pediatric study of 147 patients with HT and 144 controls, Munteanu et al. found higher neutrophil and lymphocyte counts and higher CBC-derived indices in HT (all P<0.001). NLR showed the strongest association with diagnosis, but indices did not differ among HT functional subgroups. These results are informative for pathobiology but should not be transferred directly to adult practice because hematological reference patterns, pubertal status, disease duration, and comorbidities differ. [21]

Additional regional evidence continues to reproduce the discordance. A 2025 cross-sectional study of 100 adults with hypothyroid HT and 50 controls reported higher NLR (2.1 ± 0.6 vs 1.77 ± 1.3) but lower PLR on the study's reported scale (8.6 ± 4.9 vs 11.2 ± 2.9). TSH correlated weakly with NLR (r=0.220, P=0.033), whereas TPOAb did not. Variability in ratio scaling and reporting itself illustrates the need for harmonized analytical conventions. [22]

Table 1. Principal clinical studies evaluating NLR and PLR in Hashimoto thyroiditis

Study	Material and design	Principal findings	Main limitation
Bilge et al.[12]	145 euthyroid women with HT; 60 age-matched controls	NLR: 2.29±0.65 vs 1.68±0.40; PLR: 164.95±55.14 vs 106.88±32.19 (both P<0.001).	Retrospective, female-only cohort; wide overlap and no externally validated threshold.
Aktas et al.[13]	90 patients with HT; 64 healthy controls	Median NLR was 2.1 (1.3–5.8) vs 1.9 (0.6–3.3), P=0.04.	Small absolute difference with extensive distributional overlap; no diagnostic validation.
Onalan and Donder[15]	121 overt/subclinical hypothyroid HT patients; 100 controls	NLR was higher (2.43±0.94 vs 2.11±0.81), but PLR was lower (130.8±50.5 vs 145.3±58.5).	Retrospective mixed functional states; neither ratio correlated significantly with TSH, FT4, or TPOAb.
Resber et al.[16]	93 euthyroid HT patients; 86 controls from an obesity clinic	NLR and PLR were numerically higher but nonsignificant (P=0.427 and P=0.089); treatment groups were similar.	Obesity-clinic selection and retrospective design limit generalizability.
Erge et al.[17]	Euthyroid HT, hypothyroid-thyrotoxic HT, and controls	Median PLR: 137, 177, and 103, respectively (P<0.001); PLR correlated positively with CRP.	Retrospective grouping; PLR alone cannot separate autoimmune activity from thyroid dysfunction.

Study	Material and design	Principal findings	Main limitation
Erinc et al.[18]	166 euthyroid HT, 91 hypothyroid HT, 58 nonimmune hypothyroid, 190 controls	SII and PLR differed from controls; NLR did not reach overall significance (P=0.059). Ratios did not separate patient subgroups.	Single-center retrospective data; autoimmune and hormone-deficiency effects remained difficult to disentangle.
Bozdag et al.[19]	268 HT patients across three functional states; 124 controls	NLR was higher in all HT groups; PLR was lower overall. Neither index distinguished functional states.	Retrospective design; hyperthyroid HT subgroup and heterogeneous treatment status complicate interpretation.
Demir and Tastemur[20]	92 patients with HT; 92 controls; multivariable and ROC analyses	NLR, PLR, and SII were nonsignificant; ESR, monocytes, hemoglobin, and LMR differed. LMR and FT4/TSH were independent predictors.	Retrospective single-center study; antibody AUC estimates were affected by incorporation bias.
Munteanu et al.[21]	147 pediatric HT patients; 144 controls	CBC-derived indices were higher in HT (P<0.001); NLR had the strongest diagnostic association but did not separate functional subgroups.	Pediatric findings are not directly transferable to adults; retrospective design.
Murad et al.[22]	100 adults with hypothyroid HT; 50 controls	NLR was higher (2.1±0.6 vs 1.77±1.3), whereas PLR was lower on the reported scale (8.6±4.9 vs 11.2±2.9).	Cross-sectional design and unusual PLR scaling limit comparison with other cohorts.

Abbreviations: CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; FT4, free thyroxine; HT, Hashimoto thyroiditis; LMR, lymphocyte-to-monocyte ratio; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; ROC, receiver operating characteristic; SII, systemic immune-inflammation index; TPOAb, thyroid peroxidase antibodies; TSH, thyroid-stimulating hormone.

Why the Results Conflict

The first source of heterogeneity is phenotype definition. Studies variously enroll antibody-positive euthyroid patients, subclinical hypothyroidism, overt hypothyroidism, mixed functional states, treated patients, or patients selected from obesity and surgical clinics. TSH and FT4 reflect current function, whereas antibodies and ultrasonography reflect autoimmunity and structural change. Combining these domains can conceal distinct biological subgroups and produce apparently contradictory results. [5,16]

Control selection is equally important. Obesity, insulin resistance, dyslipidemia, smoking, periodontal disease, occult infection, menstrual iron loss, and common medications can alter neutrophils, lymphocytes, or platelets by amounts comparable to the reported HT-control differences. Retrospective databases often lack high-sensitivity CRP, ferritin, vitamin B12, folate, recent exercise, menstrual status, or detailed medication exposure. Residual confounding is therefore likely even when obvious inflammatory disease is excluded. [6,10]

Preanalytical and statistical factors also matter. CBC values change with fasting, hydration, collection time, acute stress, and analyzer characteristics. Ratios amplify measurement variation because an error or physiological shift in either component changes the final value. Most HT studies are single-center and cross-sectional, test multiple indices, and select thresholds within the same cohort. Few provide external validation, repeated measurements, decision-curve analysis, or evidence that NLR or PLR improves classification beyond TSH, FT4, antibodies, age, and body mass index. [9,20]

Finally, NLR and PLR are not equivalent biological measures. A rise in NLR with a fall in PLR may occur when neutrophils increase but platelets fall or lymphocytes remain stable. A high PLR may reflect iron-deficiency thrombocytosis rather than thyroid inflammation. Reporting only ratios without the underlying cell counts can therefore obscure the causal pattern. Each index should be decomposed into its numerator and denominator before clinical interpretation. [11,15]

Relationships With Thyroid Function, Autoantibodies, and Conventional Inflammation

Correlations between NLR or PLR and TSH, FT4, TPOAb, or TgAb have generally been weak or inconsistent. This is biologically plausible: TSH measures pituitary compensation, FT4 measures

hormone availability, antibodies indicate immune recognition, and CBC ratios reflect circulating cell balance. These variables occupy different positions in the disease pathway and need not change in parallel. A high antibody concentration can coexist with euthyroidism and a normal NLR, while severe hypothyroidism can influence hematopoiesis without indicating greater antibody-mediated activity. [4,8]

CRP and ESR may offer a more direct, although still nonspecific, signal of systemic inflammation. Some studies found positive relationships between CRP and PLR or NLR, whereas others identified ESR, monocyte count, or LMR as more informative. The 2026 adult analysis found that NLR and PLR were unremarkable despite higher ESR and altered LMR, reinforcing that failure of a CBC ratio to rise does not exclude low-grade systemic inflammation. [17,20]

Broader composite indices such as systemic immune-inflammation index, calculated from platelets, neutrophils, and lymphocytes, may capture additional information but inherit the same confounders. Population data have linked systemic immune-inflammation index with thyroid function, yet causality cannot be inferred and obesity substantially modifies the association. Adding more mathematical combinations does not solve weak biological specificity unless the new index is prospectively validated against a clinically meaningful outcome. [19,23]

Effects of Thyroid Functional State and Treatment

The available evidence does not support a consistent stepwise increase in NLR or PLR from euthyroidism to subclinical and overt hypothyroidism. Several studies observed differences between HT and controls but no separation among functional subgroups. This suggests that any systemic immune signal may be related more to the autoimmune background or host phenotype than to the current serum hormone category. It also challenges the use of these ratios as surrogates for biochemical severity. [18,19]

Levothyroxine corrects hormone deficiency and remains the standard treatment for hypothyroidism, but it is not an immunosuppressive therapy. Studies comparing treated and untreated euthyroid HT patients found no consistent NLR or PLR difference. This does not prove persistent harmful inflammation; treatment allocation, duration, achieved TSH, adherence, and baseline disease severity are confounded in retrospective comparisons. Longitudinal within-patient studies before and after stable biochemical control are required. [16,24]

Clinical Interpretation

NLR and PLR should not be used to establish or exclude HT. Their distributions overlap widely with healthy individuals and with other inflammatory, metabolic, infectious, and neoplastic conditions. They cannot replace TSH, FT4, TPOAb, TgAb, clinical assessment, or ultrasonography. Both ratios may be reported as contextual information when a CBC is already available, but an unexpected elevation should trigger review for common confounders rather than automatic attribution to thyroid autoimmunity. [5,6]

The ratios are also unsuitable for distinguishing HT from papillary thyroid carcinoma or other thyroid inflammation. A comparative study found NLR increased in thyroiditis and PLR increased in both thyroiditis and papillary carcinoma, with insufficient separation between inflammatory and malignant groups. Thyroid-nodule risk assessment must continue to rely on ultrasonographic features, cytology when indicated, and established clinical algorithms. [25]

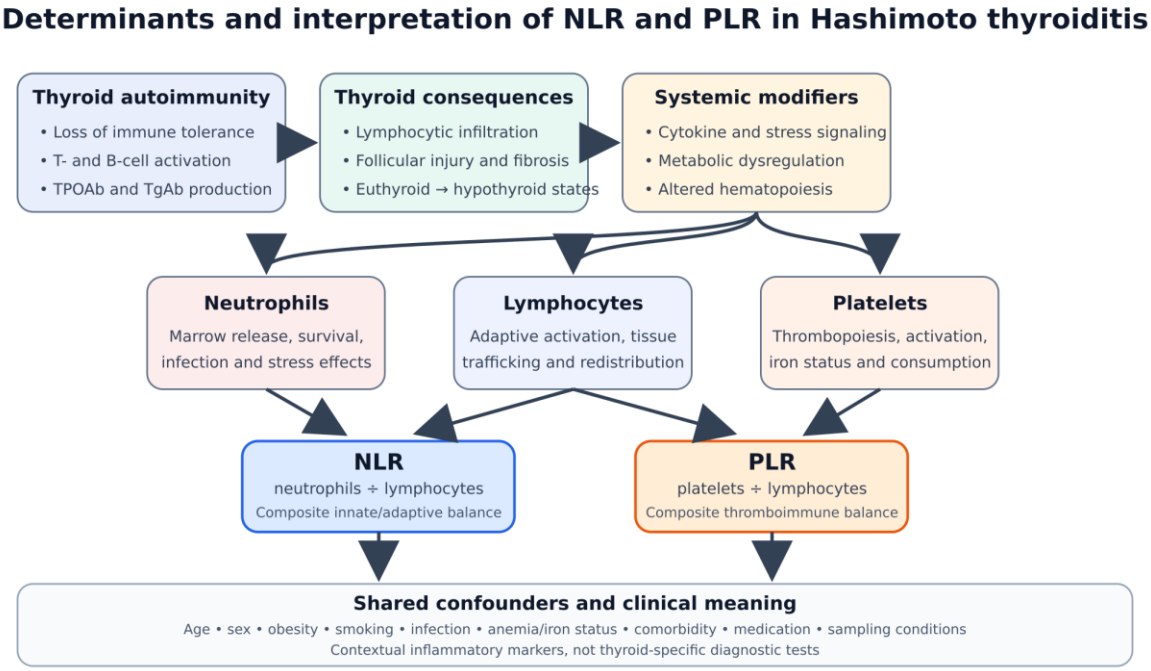
In Egyptian practice, CBC-derived ratios are appealing because they are inexpensive and widely available. Recent Egyptian data have expanded understanding of HT phenotypes and ultrasonographic findings, but local reference distributions and prospective validation of NLR and PLR remain limited. Any proposed Egyptian threshold should therefore be derived in a representative multicenter cohort and validated externally before clinical adoption. [26]

Research Priorities

Future studies should recruit clearly defined euthyroid, subclinical, and overt hypothyroid HT groups; include nonimmune hypothyroidism and healthy controls; and prespecify clinically meaningful outcomes. Repeated fasting morning CBC measurements should be obtained on the same analyzer alongside TSH, FT4, TPOAb, TgAb, CRP, ESR, ferritin, iron indices, vitamin B12, folate, lipid profile, glycemic measures, renal and hepatic function, body composition, smoking, infection screening, and medication exposure. [20,27]

Analysis should retain neutrophil, lymphocyte, and platelet counts rather than considering ratios alone. Multivariable models should test whether NLR or PLR improves discrimination, calibration, and decision-making beyond standard thyroid variables. External validation, sex-specific and age-specific analysis, and longitudinal assessment after stable levothyroxine treatment are essential. Immunophenotyping and cytokine studies could then determine whether a particular CBC pattern corresponds to Th1/Th17 activity, regulatory T-cell dysfunction, metabolic inflammation, or another systemic phenotype. [7,28]

Figure 1. Biological and clinical determinants of NLR and PLR in Hashimoto thyroiditis



The proposed model separates thyroid autoimmunity from the nonthyroidal determinants of circulating blood cells. Local lymphocytic thyroid injury may contribute to systemic cytokine signaling, neutrophil recruitment, lymphocyte redistribution, and platelet activation, while hypothyroidism adds metabolic and hematopoietic effects. Age, obesity, smoking, infection, anemia, nutritional status, comorbid disease, medication, and sampling conditions act on the same cell counts. The resulting NLR and PLR are therefore composite contextual markers rather than thyroid-specific measurements. [3,9]

Conclusion

NLR and PLR provide a biologically reasonable, inexpensive view of circulating immune-cell balance in Hashimoto thyroiditis, but their clinical meaning is constrained by substantial overlap, confounding, and inconsistent direction of change. Current evidence supports neither a universal threshold nor independent use for diagnosis, staging, or treatment monitoring. Their most defensible role is exploratory and contextual, interpreted alongside the component cell counts, thyroid function, antibodies, conventional inflammatory markers, and the patient's metabolic and clinical profile. Standardized prospective studies must demonstrate reproducible incremental value before either ratio becomes part of routine HT decision-making.

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