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ORIGINAL RESEARCH

Laboratory Parameters Can Serve as Objective Auxiliary Tools for Assessing Disease Severity in Hidradenitis Suppurativa: A 5–Year Period Single–Center Retrospective Study

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Background: Hidradenitis suppurativa (HS) is a chronic, recurrent inflammatory skin disorder. Laboratory parameters may serve as an assessment tool, providing a potential objective window into disease activity.

Objective: To systematically evaluate the utility of commonly obtained laboratory parameters in the objective assessment of disease severity in patients with HS.

Methods: This single-center retrospective study included patients who were clinically diagnosed with HS, acne inversa, or follicular occlusion triad at Peking Union Medical College Hospital between January 1, 2020, and July 1, 2025. Study data were extracted from clinical examination records and corresponding laboratory test results. Ordinal logistic regression models were constructed to evaluate the associations between laboratory parameters and Hurley stages. Heterogeneity analyses were performed to assess the consistency of these associations across anatomical regions.

Results: The study included 1750 clinical visits from 583 HS patients, with a male predominance (85.7%) and a mean age of 31.15 ±12.11 years. By systematically comparing laboratory parameters across Hurley stages, this study identified significant differences in ten markers, including white blood cell (WBC), neutrophil ratio (NEU%), platelet (PLT), plateletcrit (PCT), mean platelet volume (MPV), hemoglobin (Hb), gamma-glutamyl transferase (GGT), high-density lipoprotein cholesterol (HDL-C), erythrocyte sedimentation rate (ESR), and high-sensitivity C-reactive protein (hsCRP). Ordinal logistic regression analyses demonstrated that WBC, NEU%, PLT, PCT, platelet distribution width (PDW), GGT, ESR, and hsCRP were significantly and positively associated with increasing Hurley stage, while Hb, MPV, and HDL-C exhibited inverse associations. Heterogeneity analysis found that systemic inflammation-related markers (WBC, PDW, ESR, and hsCRP) demonstrated highly consistent associations with HS severity across anatomical locations, while metabolic parameters (HDL-C, triglycerides (TG), alanine aminotransferase (ALT), and aspartate aminotransferase (AST)) exhibited significant site-specific heterogeneity.

Conclusion: This study supports the use of readily available laboratory markers as an objective adjunct to conventional clinical severity assessments, while highlighting the anatomical specificity of HS pathophysiology.

Keywords: hidradenitis suppurativa, laboratory parameters, disease severity

Introduction

Hidradenitis suppurativa (HS) is a chronic, recurrent inflammatory skin disorder that profoundly impairs patients' quality of life. It is clinically characterized by painful, recurrent nodules, abscesses, sinus tract formation, and progressive

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scarring, predominantly affecting intertriginous areas rich in apocrine glands.[1–4] Despite increasing clinical recognition, the pathogenesis of HS remains incompletely elucidated. Although affected lesions may show secondary bacterial colonization or infection, the role of microorganisms in HS pathogenesis remains unclear.[5,6] Instead, HS is thought to involve a complex interplay of genetic susceptibility and immune dysregulation.[7–10] Currently, assessment of disease severity in HS relies primarily on clinical scoring systems, most notably the Hurley staging system and the International Hidradenitis Suppurativa Severity Score System (IHS4).[11] However, these instruments are inherently subjective and may lack sufficient sensitivity to capture subtle or dynamic changes in disease activity.[12] High-frequency skin ultrasound has emerged as a valuable modality for providing objective, quantitative measures of HS severity; nevertheless, its application is limited by restricted availability in primary care settings and relatively high cost.[13–15]

Since the pathogenic mechanisms of HS are accompanied by systemic inflammatory alterations that may be captured through routinely measured laboratory biomarkers, conventional laboratory indicators may serve as an assessment tool, providing a potential objective window into disease activity. Unlike emerging molecular or cytokine-based biomarkers, routine laboratory parameters, including complete blood count (CBC), liver and renal function tests, erythrocyte sedimentation rate (ESR), and high-sensitivity C-reactive protein (hsCRP), are widely available, cost-effective, and easily integrated into routine clinical practice. Despite these advantages, the extent to which such laboratory parameters can function as objective adjunctive tools for assessing HS severity or for dynamically monitoring disease activity over time remains insufficiently defined. Therefore, the aim of this study was to systematically evaluate the utility of commonly obtained laboratory parameters in the objective assessment of disease severity in patients with HS. By integrating severity staging with site-specific heterogeneity analyses, this study seeks to delineate both global and anatomical dimensions of laboratory markers utility in HS.

Materials and Methods

Study Design and Patients

This single-center retrospective study included patients who were clinically diagnosed with HS, acne inversa, or follicular occlusion triad at Peking Union Medical College Hospital between January 1, 2020, and July 1, 2025. Clinical diagnoses were established based on characteristic cutaneous manifestations, including recurrent nodules and cysts, scarring, and sinus tract formation, in conjunction with typical anatomical distributions such as the axillae, groin, buttocks, and genital region. The study was conducted in accordance with the principles of the Declaration of Helsinki and received approval from the institutional ethics committee (approval number I-25PJ2964).

Given the chronic, relapsing course of HS, patients frequently presented for care at multiple time points corresponding to varying levels of disease activity, during which both clinical severity and laboratory parameters could change substantially. Accordingly, clinical encounters rather than unique patients were designated as the primary unit of analysis, with each visit representing an independent assessment of disease severity and associated laboratory findings at that specific time point. This visit-based analytical framework enabled a more comprehensive characterization of disease severity and its dynamic evolution across the disease course. Repeated visits from the same patient were used to describe temporal disease characteristics and were not intended for individual-level causal inference. To mitigate potential confounding related to repeated measurements, demographic variables including age and sex were adjusted for in the regression analyses.

Data Collection

Study data were extracted from clinical examination records and corresponding laboratory test results. All clinical assessments were performed by dermatologists. Collected variables included sex, age at disease onset, family history of HS, smoking and alcohol consumption history, height, weight, body mass index (BMI), anatomical distribution of lesions, and disease severity as assessed by the Hurley staging system and the International Hidradenitis Suppurativa Severity Score System (IHS4). Laboratory parameters comprised CBC, liver and renal function tests, ESR, and hsCRP.

Statistical Analysis

Categorical variables were summarized as frequencies and percentages. Continuous variables were assessed for normality using the Shapiro–Wilk test. Variables with a normal distribution were expressed as mean and standard deviation, whereas non-normally distributed variables were summarized using median and interquartile range. For comparisons across Hurley stages, continuous variables were analyzed using one-way analysis of variance when assumptions of normality and homogeneity of variance were met; otherwise, the Kruskal–Wallis test was applied. Differences in categorical variables between groups were evaluated using the chi-square test, with Fisher's exact test used when expected cell counts were small.

Ordinal logistic regression models were constructed to evaluate the associations between laboratory parameters and disease severity, with Hurley stage specified as the ordered dependent variable and laboratory indicators entered as independent variables. Age and sex were included as covariates to account for potential confounding effects. Model outputs included regression coefficients, standard errors, Wald statistics, *P* values, odds ratios (ORs), and corresponding 95% confidence intervals (CIs), allowing assessment of the independent contribution of each laboratory parameter to disease severity. To assess the consistency of these associations across different anatomical regions, heterogeneity analyses were performed. Separate site-specific ordinal logistic regression models were generated for each affected anatomical site, and ORs with 95% CIs were compared to evaluate potential site-specific variability in effect estimates.

As expected in a retrospective study, missing data were present, resulting in variable effective sample sizes across different analyses. Accordingly, all statistical analyses were conducted using the actual number of available observations for each variable.

Results

Demographic Characteristics

A total of 1750 clinical visits from 583 patients were included in the analysis. Among these patients, 85.7% were male and 14.3% were female. The mean age was 31.15±12.11 years. Mean height, weight, and body mass index (BMI) were 176.63±6.9 cm, 92.83±20.4 kg, and 29.49±9.44, respectively. With respect to lifestyle and familial factors, 57.2% of patients reported a history of smoking, 35.2% reported alcohol consumption, and 36.8% reported a family history of HS.

Clinical Characteristics of HS

Across the included clinical visits, 71.7% were diagnosed as HS, 29.6% as follicular occlusion triad, and 1.3% as acne inversa. Based on disease severity assessment, 30.2%, 56.3%, and 13.5% of visits corresponded to Hurley stages 1, 2, and 3, respectively. The axillae were the most frequently involved anatomical sites, with left and right axillary involvement observed in 69.4% (1215 visits) and 69.6% (1218 visits), respectively (Figure 1). The head represented the second most commonly affected region, accounting for 61.2% of visits (1068 visits). Involvement of the groin and buttocks was also common, with the left and right groin affected in 33.2% (581 visits) and 32.9% (576 visits), and the left and right buttocks in 33.1% (579 visits) and 35.1% (614 visits), respectively. The neck and genital regions were less frequently involved, with prevalence rates of 18.9% (330 visits) and 8.3% (145 visits), respectively. Additionally, involvement of other anatomical sites was reported in approximately 50.7% of visits.

BMI and Laboratory Parameters Across Hurley Stages

BMI was significantly higher in patients with Hurley stages 2 and 3 compared with those classified as stage 1 (all $P < 0.01$), while patients with stage 3 disease exhibited a slightly lower BMI than those with stage 2 disease (Table 1).

With respect to CBC parameters, white blood cell (WBC) count, neutrophil ratio (NEU%), platelet (PLT) count, and plateletcrit (PCT) were significantly elevated in patients with Hurley stage 3 compared with those in stages 1 and 2 (all $P < 0.01$). Notably, WBC demonstrated a progressive increase with advancing Hurley stage. In contrast, hemoglobin (Hb) levels and mean platelet volume (MPV) were significantly reduced in stage 3 patients relative to stages 1 and 2 (all $P < 0.01$), with MPV showing a decreasing trend across increasing disease severity.

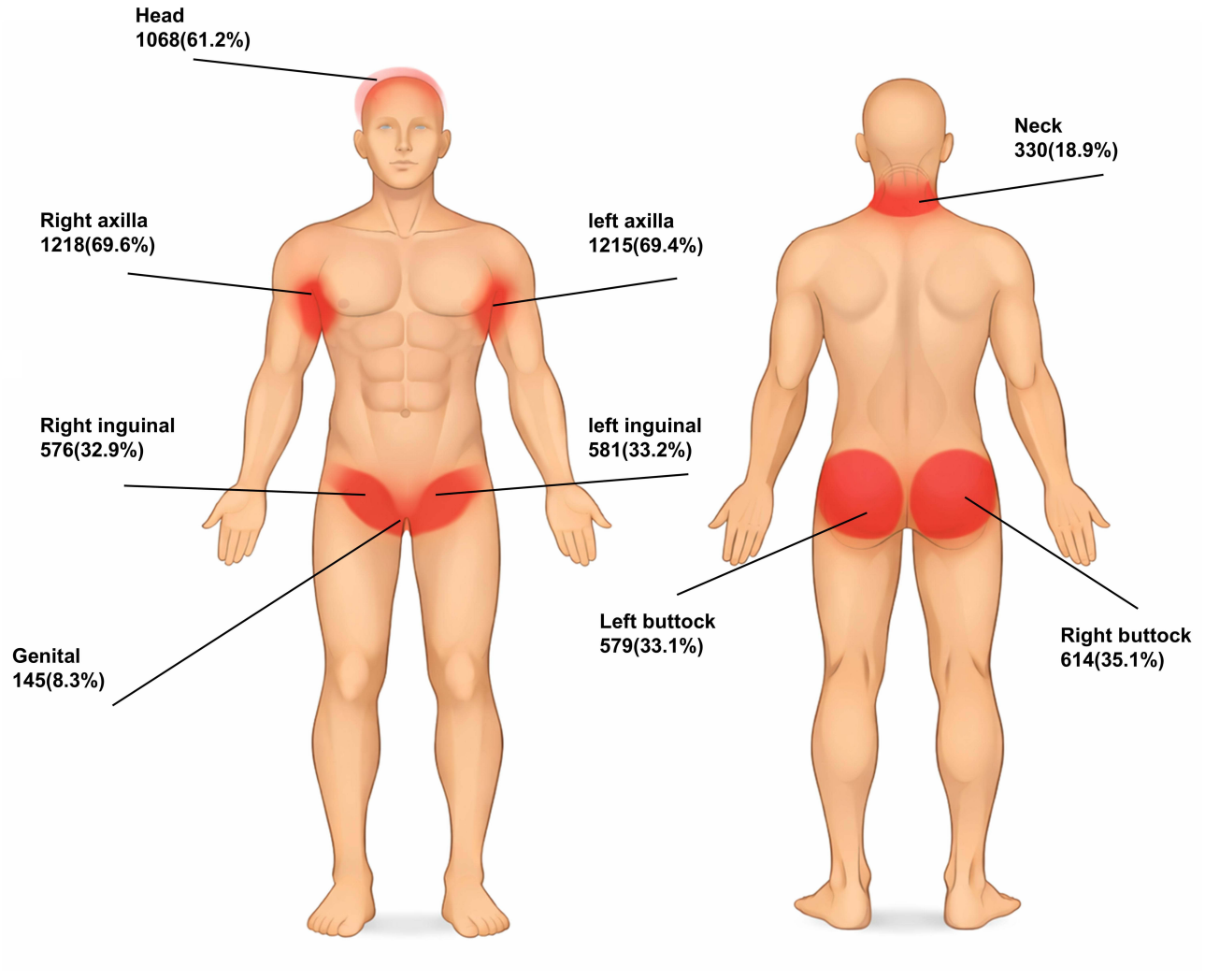

Figure 1 The Distribution of Affected Sites in Patients with HS. Axillary involvement was observed in 69.4% (left, 1215 visits) and 69.6% (right, 1218 visits). Head involvement occurred in 61.2% (1068 visits). The groin was affected in 33.2% (left, 581 visits) and 32.9% (right, 576 visits), and the buttocks in 33.1% (left, 579 visits) and 35.1% (right, 614 visits). The neck was involved in 18.9% (330 visits), and genital involvement was observed in 8.3% (145 visits). Involvement of other anatomical sites was reported in approximately 50.7% of visits.

Among biochemical parameters, gamma-glutamyl transferase (GGT) levels were significantly higher in patients with Hurley stage 3 compared with those in stages 1 and 2 (all $P < 0.01$), exhibiting a positive trend with increasing disease severity. Conversely, high-density lipoprotein cholesterol (HDL-C) levels were significantly lower in patients with stage 3 disease (all $P < 0.01$).

Regarding inflammatory markers, both ESR and hsCRP were significantly elevated in patients with Hurley stage 3 compared with stages 1 and 2. Furthermore, hsCRP demonstrated a stepwise increase with advancing Hurley stage (Table 1).

Association Between Laboratory Parameters and Hurley Stage

Ordinal logistic regression analyses were performed to evaluate the associations between laboratory parameters and disease severity in patients with HS, with Hurley stage specified as the ordered dependent variable and laboratory indices entered as independent variables. All models were adjusted for potential confounding effects of age and sex (Table 2 and Figure 2).

Among CBC parameters, WBC, NEU%, PLT, PCT, and platelet distribution width (PDW) were significantly and positively associated with increasing Hurley stage. In contrast, Hb concentration and MPV demonstrated significant

Table 1 The BMI and Laboratory Parameters Across Hurley Stages

Variables	Hurley Stage 1 [MEAN±SD or M[Q1, Q3]	Hurley Stage 2 [MEAN±SD or M[Q1, Q3]	Hurley Stage 3 [MEAN±SD or M[Q1, Q3]	P-values
BMI	21.60 (21.60, 56.46)	33.91 (29.07, 35.02)	26.83 (23.37, 29.40)	<0.001[a]
WBC (10^9/L)	8.62 (7.33, 10.51)	9.02 (7.40, 10.17)	11.46 (9.76, 12.66)	<0.001[a]
NEU (%)	69.50 (59.50, 75.55)	64.65 (57.32, 71.45)	72.70 (67.30, 76.70)	<0.001[a]
Hb (g/L)	147.50 (139.00, 153.00)	148.00 (133.00, 158.00)	134.50 (115.25, 151.50)	0.0051[a]
PLT (10^9/L)	288.50 (250.25, 328.75)	281.00 (243.00, 329.25)	371.00 (291.50, 443.50)	<0.001[a]
PCT (%)	0.29 (0.25, 0.31)	0.28 (0.24, 0.33)	0.31 (0.27, 0.42)	<0.001[a]
PDW (fl)	10.35 (9.83, 11.47)	11.00 (10.00, 12.45)	10.80 (10.00, 27.10)	0.283
MPV (fl)	9.75 (9.40, 10.40)	9.70 (9.20, 10.40)	9.40 (8.80, 9.60)	<0.001[a]
P-LCR (%)	22.30 (19.65, 27.88)	22.70 (19.40, 28.00)	21.15 (19.02, 23.52)	0.0581[b]
ALT (U/L)	16.00 (11.00, 36.00)	22.00 (14.00, 39.00)	21.00 (11.00, 43.00)	0.288
AST (U/L)	17.00 (14.00, 22.00)	19.00 (15.00, 23.00)	20.00 (14.00, 25.00)	0.575
GGT (U/L)	20.00 (17.50, 25.00)	34.50 (23.25, 60.75)	50.00 (35.25, 67.50)	<0.001[a]
Cr (mmol/L)	0.07 (0.06, 0.08)	0.07 (0.06, 0.08)	0.07 (0.06, 0.08)	0.134
Glu (mmol/L)	4.90 (4.70, 5.12)	5.10 (4.70, 5.50)	4.90 (4.50, 5.45)	0.222
UA (μmol/L)	415.61 ± 110.95	411.55 ± 110.27	421.10 ± 81.66	0.892
TG (mmol/L)	0.93 (0.66, 1.18)	1.11 (0.91, 1.71)	1.14 (0.89, 1.47)	0.066
TC (mmol/L)	4.05 (3.67, 4.35)	4.33 (3.96, 5.16)	4.46 (3.47, 4.83)	0.100
HDL-C (mmol/L)	1.03 ± 0.22	1.08 ± 0.23	0.83 ± 0.19	<0.001[a]
LDL-C (mmol/L)	2.54 (2.24, 3.08)	2.64 (2.33, 3.29)	2.91 (2.44, 3.27)	0.453
ESR	20.00 (17.00, 24.25)	12.00 (4.00, 23.50)	41.50 (29.25, 67.25)	0.0011[a]
hsCRP	4.13 (1.29, 19.03)	5.13 (2.41, 22.53)	37.88 (20.33, 56.24)	<0.001[a]

Notes: In this table, the Shapiro–Wilk test was used for assessed normality for the continuous variables, variables that followed a normal distribution are presented as mean ± standard deviation (Mean ± SD), variables that did not conform to a normal distribution are expressed as median with interquartile range [M (Q1, Q3)], and group comparisons were conducted using the Kruskal–Wallis *H*-test or one-way ANOVA, as appropriate. [a] indicates the p-value<0.01; [b] indicates the p-value<0.05.

Abbreviations: BMI, body mass index; WBC, white blood cell count; NEU%, neutrophil ratio; Hb, hemoglobin; PLT, platelet; PCT, plateletcrit; PDW, platelet distribution width; MPV, mean platelet volume; P-LCR, platelet-large cell ratio; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GGT, gamma-glutamyl transferase; Cr, creatinine; Glu, glucose; UA, uric acid; TG, triglycerides; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; ESR, erythrocyte sedimentation rate; hsCRP, high-sensitivity C-reactive protein.

inverse associations with disease severity. With the exception of PCT, the 95% confidence intervals (CIs) for these parameters were relatively narrow, indicating good model stability and precision. Notably, PCT yielded extremely high odds ratio (OR) estimates in certain models, accompanied by substantial widening of the corresponding 95% CIs, suggesting potential influence from skewed data distributions or quasi-complete separation.

Among biochemical parameters, GGT showed a significant positive association with Hurley stage. Conversely, high-density lipoprotein cholesterol (HDL-C) was significantly negatively associated with disease severity; however, the relatively wide 95% CI indicated greater interindividual variability in its predictive effect.

Both ESR and hsCRP exhibited significant positive associations with increasing Hurley stages. Notably, the 95% CI for hsCRP was highly concentrated, reflecting a particularly consistent and robust association with disease severity across the study population. No statistically significant associations were observed between the remaining laboratory parameters and Hurley stages (Table 2 and Figure 2).

Heterogeneity of Associations Across Anatomical Sites

To further assess the consistency of these associations across different anatomical regions, subsite-specific heterogeneity analyses were conducted using separate ordinal logistic regression models for each affected site (Tables 3 and 4).

Systemic inflammation-related markers, including WBC, ESR, and hsCRP, demonstrated odds ratios greater than 1 in the vast majority of site-specific models, with 95% CIs that did not cross 1 and remained relatively narrow and

Table 2 Ordinal Logistic Regression Analysis on the Association Between Laboratory Parameters and Hurley Stage

Indicator	Coefficient	Standard Deviation	Waldχ^2	P-values	OR	OR 95% CI Lower Bound	OR 95% CI Upper Bound
WBC	0.1578	0.0458	11.8710	0.0006[a]	1.1710	1.0704	1.2810
NEU%	0.0340	0.0121	7.8560	0.0051[a]	1.0346	1.0103	1.0595
Hb	−0.0305	0.0073	17.5502	<0.0001[a]	0.9700	0.9563	0.9839
PLT	0.0086	0.0017	26.9231	<0.0001[a]	1.0087	1.0054	1.0120
PCT	7.1133	1.7637	16.2654	0.0001[a]	1228.1556	38.7212	38,954.5636
PDW	0.0382	0.0123	9.6162	0.0019[a]	1.0389	1.0141	1.0643
MPV	−0.6617	0.1603	17.0420	<0.0001[a]	0.5160	0.3768	0.7064
P-LCR	−0.0431	0.0255	2.8633	0.0906	0.9578	0.9112	1.0068
ALT	−0.0051	0.0040	1.6179	0.2034	0.9949	0.9871	1.0028
AST	−0.0087	0.0131	0.4386	0.5078	0.9913	0.9662	1.0172
GGT	0.0146	0.0063	5.3536	0.0207[b]	1.0147	1.0022	1.0273
Cr	−1.6641	2.4460	0.4629	0.4963	0.1894	0.0016	22.8726
Glu	0.0965	0.1003	0.9255	0.3360	1.1013	0.9047	1.3407
UA	−0.0013	0.0017	0.5327	0.4655	0.9987	0.9954	1.0021
TG	0.0457	0.0789	0.3354	0.5625	1.0468	0.8967	1.2219
TC	−0.0003	0.0049	0.0027	0.9589	0.9997	0.9902	1.0094
HDL-C	−2.7457	0.8000	11.7796	0.0006[a]	0.0642	0.0134	0.3080
LDL-C	0.1072	0.2155	0.2474	0.6189	1.1132	0.7296	1.6983
ESR	0.0511	0.0146	12.2518	0.0005[a]	1.0524	1.0227	1.0829
hsCRP	0.0198	0.0064	9.7014	0.0018[a]	1.0200	1.0074	1.0328

Notes: The model was adjusted for potential confounders such as sex and age. [a] indicates the p-value<0.01; [b] indicates the p-value<0.05.

Abbreviations: OR, odds ratio; CI, confidence interval; WBC, white blood cell count; NEU%, neutrophil ratio; Hb, hemoglobin; PLT, platelet; PCT, plateletcrit; PDW, platelet distribution width; MPV, mean platelet volume; P-LCR, platelet-large cell ratio; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GGT, gamma-glutamyl transferase; Cr, creatinine; Glu, glucose; UA, uric acid; TG, triglycerides; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; ESR, erythrocyte sedimentation rate; hsCRP, high-sensitivity C-reactive protein.

concentrated. These findings indicate a highly stable positive association between systemic inflammatory burden and Hurley stage that was largely consistent across anatomical sites. Platelet distribution width also showed a significant positive association in most subsite-specific models, with similarly concentrated confidence intervals.

In contrast, metabolism-related parameters displayed pronounced heterogeneity in their associations with Hurley stage across different anatomical sites. HDL-C exhibited a significant protective association (OR < 1) in models for all sites except the right buttock, where both the direction and statistical significance of the association were inconsistent. Additionally, triglycerides (TG), alanine aminotransferase (ALT), and aspartate aminotransferase (AST) demonstrated variable levels of statistical significance and substantially wider 95% CIs across site-specific models compared with inflammatory markers. Total cholesterol showed a statistically significant association with Hurley stage only in the genital region. Collectively, these findings suggest that the predictive value of metabolism-related parameters is more susceptible to modulation by local tissue microenvironmental differences.

Consistent with the overall model findings, PCT yielded extremely high OR estimates in certain site-specific analyses, with confidence intervals expanding by an order of magnitude. This pattern indicates that effect estimation for PCT may be influenced by highly skewed distributions, extreme values, or quasi-complete separation. Although statistical significance was achieved in some models, the limited statistical power of this parameter suggests that its clinical interpretation should emphasize the direction of association rather than the absolute magnitude of effect.

Discussion

Based on 1750 clinical visits from 583 patients with HS, our cohort showed a marked male predominance (85.7%) and a relatively young mean age of 31.15 ± 12.11 years. A family history of HS was reported by 36.8% of patients, a proportion comparable to that observed in international cohorts, supporting the role of heritable factors in disease susceptibility.[16] In this cohort, HS was used more often than acne inversa (AI), reflecting a preference in clinical

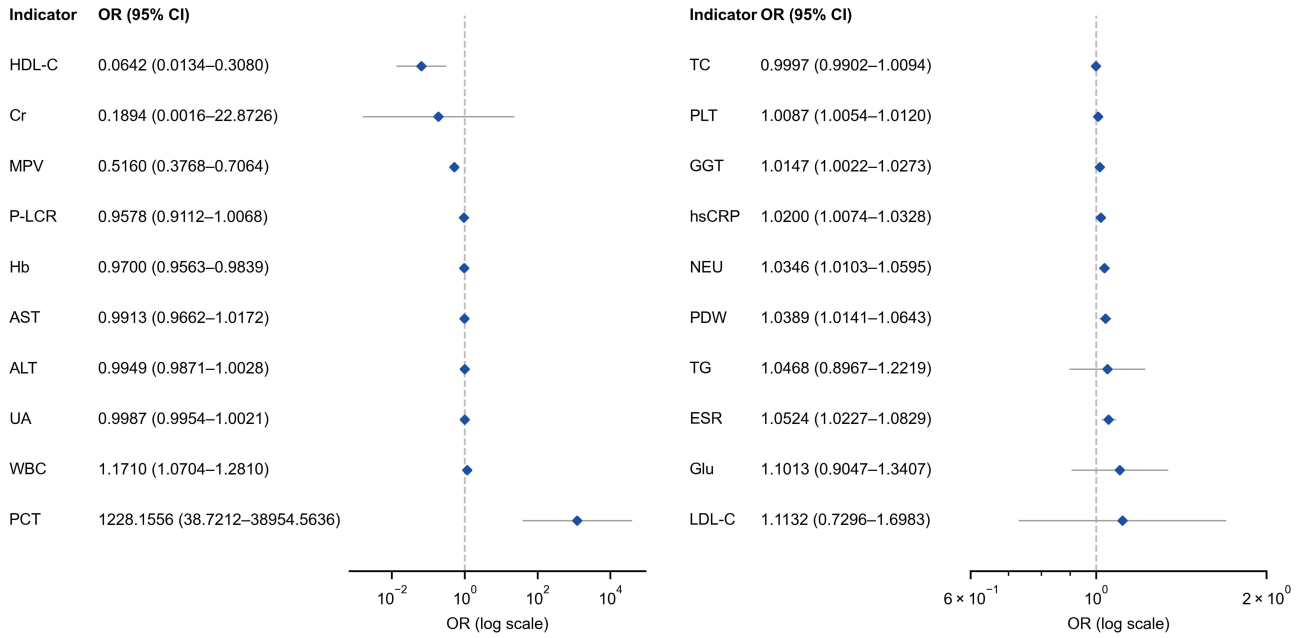

Figure 2 Association between Laboratory Parameters and Disease Severity (Hurley stage) in Hidradenitis Suppurativa.
Abbreviations: OR, odds ratio; CI, confidence interval; WBC, white blood cell count; NEU%, neutrophil ratio; Hb, hemoglobin; PLT, platelet; PCT, plateletcrit; PDW, platelet distribution width; MPV, mean platelet volume; P-LCR, platelet-large cell ratio; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GGT, gamma-glutamyl transferase; Cr, creatinine; Glu, glucose; UA, uric acid; TG, triglycerides; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; ESR, erythrocyte sedimentation rate; hsCRP, high-sensitivity C-reactive protein.

terminology among Chinese clinicians, with AI often reserved for patients with HS accompanied by facial acne conglobate. The high proportion of Hurley stage 2 and 3 visits indicates that many patients presented with already established moderate-to-severe disease. Given the role of Peking Union Medical College Hospital as a top-tier tertiary referral center in China, this pattern likely reflects substantial diagnostic delay and under-recognition of HS in primary care.[17] The cohort also showed marked clinical heterogeneity, as reflected by the anatomical distribution of lesions and the frequent presence of multisite involvement. Moreover, although obesity is a recognized factor in HS onset and progression, and the higher BMI observed in patients with Hurley stage 2 or 3 supports its association with greater disease severity, the slightly lower BMI in stage 3 than in stage 2 suggests that obesity alone is insufficient to explain progression to the most severe disease.[18–21] These observations highlight the complexity of HS and reinforce the need for objective markers to assist in severity stratification.

By systematically comparing laboratory parameters across Hurley stages, this study identified significant differences in ten markers, including WBC, NEU%, PLT, PCT, MPV, Hb, GGT, HDL-C, ESR, and hsCRP. These parameters broadly clustered into two functional categories: markers related to systemic inflammation (WBC, NEU%, PLT, PCT, MPV, ESR, and hsCRP) and markers related to metabolic status (Hb, GGT, and HDL-C). Compared with previous studies showing laboratory abnormalities in HS,[22–25] this functional stratification provided a biologically grounded framework for interpreting HS severity through the relative contributions of systemic inflammation and metabolic dysregulation. This pattern suggests that HS progression is driven by an interplay between systemic inflammatory burden and metabolic dysregulation, providing a biological basis for the increased susceptibility observed in individuals with obesity. Notably, GGT levels increased in parallel with higher Hurley stages. While elevated GGT is commonly associated with obesity, it may also reflect cumulative medication exposure and hepatic metabolic stress in patients with more severe disease.

To elucidate the associations between laboratory parameters and disease severity, ordinal logistic regression analyses were performed, demonstrating that WBC, NEU%, PLT, PCT, PDW, GGT, ESR, and hsCRP were all significantly and positively associated with increasing Hurley stage. In contrast, Hb, MPV, and HDL-C exhibited significant inverse associations with disease severity. With the exception of PCT and HDL-C, which showed greater interindividual variability in their predictive effects, the remaining markers demonstrated a high degree of consistency and robustness

Table 3 Heterogeneity Analysis on the Association Between Laboratory Parameters and Hurley Stage Across Head, Neck, Axilla, and Left Inguinal

Indicators	Head	Neck	Left Axilla	Right Axilla	Left Inguinal Region
HDL-C	0.221[a]	0.022[a]	0.044[a]	0.034[a]	0.056[a]
	[0.05–0.98]	[0.00–0.22]	[0.01–0.21]	[0.01–0.17]	[0.01–0.33]
LDL-C	1.277	1.034	1.054	1.193	1.064
	[0.85–1.93]	[0.61–1.75]	[0.72–1.55]	[0.81–1.76]	[0.70–1.62]
ALT	0.997	0.976[a]	0.997	0.998	0.999
	[0.99–1.00]	[0.96–1.00]	[0.99–1.00]	[0.99–1.00]	[0.99–1.01]
NEU%	1.056[a]	1.025	1.012	1.016	1.025[b]
	[1.03–1.08]	[0.99–1.06]	[0.99–1.03]	[0.99–1.04]	[1.00–1.05]
P-LCR	0.982	0.981	0.993	0.974	0.969
	[0.94–1.03]	[0.91–1.06]	[0.95–1.04]	[0.93–1.02]	[0.92–1.02]
AST	0.992	0.971	1.002	1.007	0.996
	[0.97–1.01]	[0.92–1.02]	[0.98–1.03]	[0.98–1.03]	[0.97–1.02]
UA	0.996[a]	0.997	0.998	1.002	1.000
	[0.99–1.00]	[0.99–1.00]	[0.99–1.00]	[1.00–1.00]	[1.00–1.00]
MPV	0.629[a]	0.583[b]	0.755	0.809	0.586[a]
	[0.47–0.84]	[0.38–0.90]	[0.57–1.01]	[0.60–1.08]	[0.43–0.80]
TC	1.000	0.750	1.001	1.001	1.003
	[0.99–1.01]	[0.47–1.19]	[0.99–1.01]	[0.99–1.01]	[0.99–1.01]
TG	0.914	0.773	1.081	1.117	1.088
	[0.77–1.09]	[0.45–1.33]	[0.93–1.26]	[0.95–1.32]	[0.95–1.25]
WBC	1.187[a]	1.080[b]	1.086[b]	1.121[a]	1.133[a]
	[1.09–1.30]	[1.00–1.16]	[1.01–1.17]	[1.03–1.22]	[1.04–1.23]
Cr	0.740	0.000[a]	0.000[a]	0.000[b]	0.000
	[0.01–56.65]	[0.00–0.00]	[0.00–0.00]	[0.00–0.03]	[0.00–7.24]
Glu	0.913	0.933	1.183	1.265[b]	0.909
	[0.73–1.13]	[0.69–1.26]	[0.97–1.44]	[1.04–1.54]	[0.73–1.13]
PLT	1.006[a]	1.005[a]	1.008[a]	1.008[a]	1.009[a]
	[1.00–1.01]	[1.00–1.01]	[1.01–1.01]	[1.00–1.01]	[1.01–1.01]
PDW	1.044[a]	1.046[a]	1.030[b]	1.015	1.031[a]
	[1.02–1.07]	[1.02–1.07]	[1.01–1.05]	[0.99–1.04]	[1.01–1.06]
PCT	0.910	0.949	4548.088[a]	2504.593[a]	6398.564[a]
	[0.76–1.10]	[0.72–1.25]	[136.72–151,293.46]	[68.73–91,272.62]	[171.92–238,143.66]
ESR	1.048[a]	1.031[a]	1.031[a]	1.018	1.033[a]
	[1.02–1.08]	[1.01–1.05]	[1.01–1.05]	[1.00–1.04]	[1.01–1.06]
Hb	0.966[a]	0.979[b]	0.984[b]	0.996	0.976[a]
	[0.95–0.98]	[0.96–1.00]	[0.97–1.00]	[0.98–1.01]	[0.96–0.99]
GGT	1.014[b]	1.006	1.012[b]	1.012[b]	1.011
	[1.00–1.02]	[0.99–1.02]	[1.00–1.02]	[1.00–1.02]	[1.00–1.02]
hsCRP	1.017[a]	1.020[a]	1.017[a]	1.012[b]	1.017[a]
	[1.01–1.03]	[1.01–1.03]	[1.01–1.03]	[1.00–1.02]	[1.00–1.03]

Notes: The model was adjusted for potential confounders such as sex and age. [a] indicates the p-value<0.01; [b] indicates the p-value<0.05.

in their associations with Hurley stage. These findings are broadly in line with previous reports indicating that laboratory markers of systemic inflammation are associated with HS severity. For instance, earlier studies showed that higher Hurley stages were accompanied by increased WBC, neutrophil count and lower Hb levels, while PLT and MPV were not consistently associated with Hurley stages;[22] more recently, CRP and ESR were also shown to rise with increasing Hurley severity.[24,25] Our findings reinforce the strong and stable relationship between systemic inflammatory burden and increasing disease severity. However, the attenuation of effect sizes observed for certain hematologic and metabolic

Table 4 Heterogeneity Analysis on the Association Between Laboratory Parameters and Hurley Stage Across Right Inguinal Region, Buttock, Genital Region, and Others

Indicators	Right Inguinal Region	Left Buttock	Right Buttock	Genital Region	Others
HDL-C	0.062[a]	0.149[b]	0.310	0.070[b]	0.171[b]
	[0.01–0.36]	[0.03–0.79]	[0.06–1.53]	[0.01–0.76]	[0.04–0.76]
LDL-C	1.037	0.873	0.883	0.391[a]	1.201
	[0.68–1.57]	[0.57–1.34]	[0.58–1.36]	[0.18–0.85]	[0.82–1.77]
ALT	0.999	0.975[a]	0.976[a]	1.000	0.990[b]
	[0.99–1.01]	[0.96–0.99]	[0.96–0.99]	[0.99–1.01]	[0.98–1.00]
NEU%	1.028[b]	1.034[b]	1.035[b]	1.047	1.061[a]
	[1.00–1.05]	[1.01–1.06]	[1.01–1.06]	[1.00–1.10]	[1.03–1.09]
P-LCR	0.974	0.985	0.986	1.026	0.981
	[0.93–1.02]	[0.94–1.04]	[0.94–1.04]	[0.93–1.13]	[0.94–1.03]
AST	0.998	0.953	0.959	1.004	0.980
	[0.97–1.02]	[0.91–1.00]	[0.91–1.01]	[0.98–1.03]	[0.95–1.01]
UA	1.000	0.995[b]	0.998	1.000	0.998
	[1.00–1.00]	[0.99–1.00]	[0.99–1.00]	[0.99–1.01]	[0.99–1.00]
MPV	0.602[a]	0.588[a]	0.593[a]	0.552[b]	0.650[a]
	[0.44–0.82]	[0.42–0.81]	[0.43–0.82]	[0.33–0.92]	[0.49–0.87]
TC	1.003	0.816	0.856	0.364[a]	1.002
	[0.99–1.01]	[0.57–1.18]	[0.60–1.23]	[0.19–0.70]	[0.99–1.01]
TG	1.087	1.033	1.029	0.237[a]	0.888
	[0.95–1.25]	[0.90–1.18]	[0.89–1.18]	[0.07–0.80]	[0.71–1.11]
WBC	1.145[a]	1.113[a]	1.108[b]	1.076	1.208[a]
	[1.05–1.25]	[1.03–1.21]	[1.02–1.20]	[0.97–1.19]	[1.10–1.32]
Cr	0.000	0.000	0.000	0.000	0.753
	[0.00–1684.66]	[0.00–34,971.18]	[0.00–754,260.52]	[0.00–46,918,568.83]	[0.01–51.64]
Glu	0.823	0.970	0.989	1.011	0.975
	[0.64–1.06]	[0.78–1.21]	[0.80–1.22]	[0.73–1.40]	[0.80–1.19]
PLT	1.010[a]	1.005[a]	1.004[a]	1.007[a]	1.010[a]
	[1.01–1.01]	[1.00–1.01]	[1.00–1.01]	[1.00–1.01]	[1.01–1.01]
PDW	1.031[a]	1.035[a]	1.031[b]	1.054[a]	1.037[a]
	[1.01–1.05]	[1.01–1.06]	[1.01–1.06]	[1.02–1.09]	[1.01–1.06]
PCT	7914.502[a]	108.162[a]	61.204[b]	0.968	5128.493[a]
	[207.75–301,513.53]	[3.79–3084.33]	[2.13–1760.16]	[0.74–1.26]	[175.04–150,261.02]
ESR	1.033[a]	1.042[a]	1.040[a]	1.041[a]	1.041[a]
	[1.01–1.06]	[1.02–1.07]	[1.02–1.06]	[1.01–1.07]	[1.02–1.07]
Hb	0.979[a]	0.962[a]	0.961[a]	0.971[a]	0.954[a]
	[0.97–0.99]	[0.95–0.98]	[0.95–0.98]	[0.95–0.99]	[0.94–0.97]
GGT	1.013[b]	1.007	1.008	1.004	1.013[b]
	[1.00–1.02]	[1.00–1.02]	[1.00–1.02]	[0.99–1.02]	[1.00–1.02]
hsCRP	1.015[b]	1.019[a]	1.014[b]	1.012[b]	1.019[a]
	[1.00–1.03]	[1.01–1.03]	[1.00–1.03]	[1.00–1.02]	[1.01–1.03]

Notes: The model was adjusted for potential confounders such as sex and age. [a] indicates the p-value<0.01; [b] indicates the p-value<0.05.
Abbreviations: WBC, white blood cell count; NEU%, neutrophil ratio; Hb, hemoglobin; PLT, platelet; PCT, plateletcrit; PDW, platelet distribution width; MPV, mean platelet volume; P-LCR, platelet-large cell ratio; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GGT, gamma-glutamyl transferase; Cr, creatinine; Glu, glucose; UA, uric acid; TG, triglycerides; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; ESR, erythrocyte sedimentation rate; hsCRP, high-sensitivity C-reactive protein.

markers after multivariate adjustment suggests that inter-marker correlations may partially influence their individual predictive contributions.

To further explore whether these associations varied according to anatomical site, a heterogeneity analysis was conducted using site-specific regression models. Comparison of ORs and CIs across affected regions revealed that, although several laboratory parameters were significantly associated with Hurley stage in the overall model, their CIs

crossed unity in certain site-specific analyses, indicating potential site-dependent variability in predictive performance. Overall, systemic inflammation-related markers, including WBC, PDW, ESR, and hsCRP, demonstrated highly consistent associations with HS severity across anatomical locations. This finding underscores systemic inflammation as a fundamental driver of disease progression, largely independent of lesion distribution. In contrast, metabolic parameters, including HDL-C, TG, ALT, and AST, exhibited significant heterogeneity in their associations with disease severity across different sites. The reduced stability of these associations suggests that the predictive value of metabolic markers is more strongly influenced by site-specific factors. The observed shifts in CIs across anatomical regions indicate that, against a backdrop of relatively uniform systemic inflammatory activation, the contribution of local metabolic states to disease severity varies substantially. From a clinical perspective, these findings support prioritizing systemic inflammatory markers as core indicators of disease severity across all anatomical distributions, while emphasizing that metabolic markers should be interpreted in conjunction with the specific sites of involvement to enable a more nuanced and individualized assessment.

Collectively, these results indirectly suggest the presence of site-specific pathogenic mechanisms in HS and offer a plausible explanation for the clinically observed phenomenon whereby lesions at different anatomical locations within the same patient may respond inconsistently to identical therapeutic interventions. Taken together, these observations support a data-driven hypothesis in which HS represents a systemic inflammatory–metabolic disorder, with site-specific modulation of clinical expression reflecting differential contributions of local metabolic factors. Stratifying patients based on the predominant involvement of inflammatory versus combined inflammatory–metabolic pathways, as inferred from affected anatomical sites, may ultimately inform more targeted and effective treatment strategies. Such an approach underscores the need for multidisciplinary care models integrating dermatology, endocrinology, and primary care to comprehensively address the systemic nature of HS.

Limitations

Despite the clinically relevant insights generated by this study, several limitations warrant consideration. First, the retrospective design inherently resulted in incomplete data capture, leading to variable effective sample sizes across individual analyses. This variability may have influenced the precision and robustness of certain estimates. Second, as this investigation was conducted at a single tertiary referral center, selection bias cannot be excluded. The study population is likely enriched for patients with more severe or refractory disease, which may limit the generalizability of the findings to broader HS populations, particularly those encountered in primary care or community-based settings. In addition, the male predominance observed in this cohort should be interpreted cautiously. The sex distribution of the study population may primarily reflect the referral pattern and patient characteristics of this institution, rather than the true epidemiological distribution of HS in the broader population. The influence of healthcare-seeking behavior and sociocultural factors cannot be ruled out, and these factors may have further shaped the observed sex distribution.[26,27] Moreover, although a similar pattern has been reported in China and other East Asian countries, the present data remain insufficient to support broader epidemiological inferences.[28–30] Accordingly, validation of these results in independent, multicenter cohorts and through large-scale prospective studies is required to confirm their applicability and reproducibility.

Conclusion

This study demonstrated that WBC, NEU%, PLT, MPV, PDW, Hb, GGT, HDL-C, ESR, and hsCRP represent informative laboratory markers for assessing disease severity and progression in HS. Importantly, the associations between these laboratory parameters and Hurley stage were not uniform across anatomical sites. Systemic inflammatory markers exhibited consistently strong correlations with disease severity irrespective of lesion location, whereas lipid metabolism-related indices and select platelet parameters displayed pronounced site-specific heterogeneity. Together, these findings support the use of readily available laboratory markers as an objective adjunct to conventional clinical severity assessments while highlighting the anatomical specificity of HS pathophysiology. More broadly, the results advocate for a shift toward precision medicine in HS, in which therapeutic strategies are informed not only by disease stage but also by site-specific biological profiles.

Abbreviations

HS, Hidradenitis suppurativa; WBC, White blood cell; NEU%, Neutrophil ratio; PLT, Platelet; PCT, Plateletcrit; MPV, Mean platelet volume; Hb, Hemoglobin; GGT, Gamma-glutamyl transferase; HDL-C, High-density lipoprotein cholesterol; ESR, Erythrocyte sedimentation rate; hsCRP, High-sensitivity C-reactive protein; PDW, Platelet distribution width; TG, Triglycerides; ALT, Alanine aminotransferase; AST, Aspartate aminotransferase; IHS4, International Hidradenitis Suppurativa Severity Score System; CBC, Complete blood count; BMI, Body mass index; ORs, Odds ratios; CIs, Confidence intervals; AI, Acne inversa.

Data Sharing Statement

The datasets supporting the conclusions of this article are available from Chao Wu (email: wuchao198978@163.com) on reasonable request.

Ethics Approval

This study was reviewed and approved by the institutional review board of Peking Union Medical College Hospital (Approval number: I-25PJ2964). The requirement for informed consent was waived due to the retrospective and non-interventional design of the study, which was based solely on anonymized clinical data and did not include any direct patient contact, intervention, or inclusion of identifiable personal information or images.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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