ORIGINAL ARTICLE

Correlation of inflammatory serum markers with disease severity in patients with hidradenitis suppurativa (HS)

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Background: Data regarding the association of routinely obtained serum markers of inflammation, namely C-reactive protein (CRP), white blood cell count, and neutrophil count, with disease severity of hidradenitis suppurativa (HS) according to a scoring system have not been reported to our knowledge.

Objective: We sought to evaluate these inflammatory serum markers for assessing disease severity of HS.

Methods: Medical files of 275 patients who were referred to the outpatient HS center of the Department of Dermatology, Venereology, and Allergology, Ruhr-University Bochum, in 2013 were evaluated retrospectively.

Results: CRP levels and neutrophil count significantly differed among Hurley stages I, II and III ($P < .0001$, $P = .0002$, respectively). There were significant positive correlations among CRP levels ($r = 0.496$, $P < .0001$) and neutrophil count ($r = 0.330$, $P = .0009$) with modified Hidradenitis Suppurativa Score. CRP was a significant independent predictor for Hurley stage III (odds ratio 1.077, 95% confidence interval 1.013-1.145, $P = .016$). CRP and body mass index were significant independent predictors for severe disease according to modified Hidradenitis Suppurativa Score (odds ratio 1.065, 95% confidence interval 1.015-1.117, $P = .009$; and odds ratio 1.12, 95% confidence interval 1.009-1.243, $P = .032$, respectively).

Limitations: Files were analyzed retrospectively.

Conclusion: These inflammatory markers, especially CRP, are effective for assessing the extent of disease severity and the grade of inflammation in patients with HS. (J Am Acad Dermatol http://dx.doi.org/10.1016/j.jaad.2015.08.052.)

Key words: biomarkers; C-reactive protein; hidradenitis suppurativa; inflammation; inflammatory skin disease; serum markers for inflammation.

Hidradenitis suppurativa (HS) is a chronic inflammatory skin disease characterized by the formation of multiple abscesses, nodules, and scars in the apocrine gland-bearing areas.[1,2] The most frequently affected anatomic sites are the inguinofemoral, axillary, perianal, gluteal, and submammary regions. The prevalence of HS is estimated to be 1% to 4%.[3,4] Follicular occlusion as a result of hyperkeratosis appears to play a major role in the pathogenesis of HS and can lead to occlusion

Abbreviations used:

BMI:	body mass index
CI:	confidence interval
CRP:	C-reactive protein
HS:	hidradenitis suppurativa
IL:	interleukin
IQR:	interquartile range
mHSS:	modified Hidradenitis Suppurativa Score
OR:	odds ratio
WBC:	white blood cell count

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of apocrine glands with subsequent follicular rupture and inflammation.[5] Genetic predisposition, obesity, and cigarette smoking are considered to be risk factors.[6]

One of the available clinical measures for assessing HS disease severity is the Hurley classification system, which defines 3 stages of severity.[7] Stage I is characterized by single or multiple abscesses without sinus tracts, stage II is described as recurrent abscesses with tract formation and cicatrization separated by normal-appearing skin, and stage III is defined as multiple interconnected sinus tracts without normal-appearing skin in between. A more precise HS severity score is the modified Hidradenitis Suppurativa Score (mHSS), which considers the extent of disease and inflammation by counting the number of individual nodules and fistulas.[6]

CAPSULE SUMMARY

- No data are available to our knowledge regarding the association of routinely obtained serum markers of inflammation with disease severity of hidradenitis suppurativa according to a scoring system.
- C-reactive protein and neutrophil count are effective parameters for assessing disease severity and grade of inflammation.
- These markers should be considered as a valuable extension to currently available clinical scoring systems.

Assessments of disease severity are currently based only on the grading of skin symptoms. Because of the simplicity of the Hurley staging system, it is routinely used in clinical evaluations. Although it is a useful tool, choosing between conservative and surgical treatment options, it is static, nonquantitative, and not suitable for assessing the extent of inflammation. In contrast, the more complex and dynamic mHSS is better suited to assess disease severity and grade of inflammation. However, its routine clinical use is time-consuming and usually reserved for clinical trials. Thus, an accurate disease quantification system and objective biomarkers for HS are needed. Recently, biomarkers for HS have been proposed, such as soluble interleukin (IL)-2 receptor,[8] S100A8/A9,[9] and chitinase-3-like protein 1 (YKL-40).[10] In contrast, serum markers for inflammation, namely C-reactive protein (CRP), white blood cell count (WBC), and neutrophil count, have already been established for evaluating the grade of inflammation in the clinic. Data regarding the association of routinely obtained serum markers of inflammation with disease severity of HS according to a scoring system have not been reported. Hence, we evaluated these inflammatory serum markers for assessing disease severity according to the Hurley staging system and the mHSS.

METHODS

Retrospectively, medical files of 275 patients referred to the outpatient HS/AI Center of the Department of Dermatology, Venereology, and Allergology, Ruhr-University Bochum, in 2013 were evaluated. Patients were included if diagnosis of HS was confirmed, which required the presence of the well-established criteria[11] and if mHSS and laboratory data at the time of consultation were available. Exclusion criteria were patients younger than 18 years, receiving ongoing antibiotic or immunosuppressive therapy, with a history of surgery within 4 weeks before consultation, with a suspected infectious disease or if antibiotic or immunosuppressive agents had been administered within 4 weeks before consultation. Disease severity was assessed via mHSS and Hurley stage of the most severely affected region. The study was approved by the local ethics committee and was performed according to the declaration of Helsinki.

Measurement of laboratory parameters

WBC and differential counts were determined using a fluorescent flow cytometry Sysmex XE-5000 analyzer (Sysmex, Kobe, Japan). A turbidimetric assay on Cobas 8000 (Roche Diagnostics, Mannheim, Germany) was used to measure CRP serum levels. A CRP level greater than 5 mg/L, WBC greater than $9.5 \times 10^3/\mu L$, and neutrophil count greater than $7.2 \times 10^3/\mu L$ were regarded as abnormal values.

Statistical analysis

Continuous data are presented as the mean (SD) or median (interquartile range [IQR]) and were compared using the Student t test or the nonparametric Mann-Whitney U test. Categorical data are presented as numbers (percentages) and were compared using the χ^2 or Fisher exact test.

Correlations between variables were determined via the Spearman coefficient of rank correlation. Post hoc power calculations for correlation coefficient revealed that the number of patients included in our study allowed detection of an effect size of 0.271 with 80% power and an alpha risk of 5%. Thus, the

Table I. Characteristics of 104 patients with hidradenitis suppurativa enrolled in the study

Characteristic	Total, n = 104	Men, n = 26	Women, n = 78	*P* value
Age, mean (SD), y	37.7 (11.1)	37.8 (13.2)	37.7 (10.4)	.94
History of HS				
Age at onset, mean (SD), y	25 (9.7)	27.3 (11.4)	24.3 (9.1)	.169
Disease duration, median (IQR), y	11 (6-18)	9 (5-15)	12.5 (6.5-22)	.142
Diagnostic delay, median (IQR), y	8.8 (3.7-15.1)	5.6 (3.7-11.3)	9.1 (3.9-16.5)	.242
First-degree relative with HS, n (%)	20 (19.2)	1 (3.8)	19 (24.4)	.022
Smoking				
Current smoker, n (%)	69 (66.3)	17 (65.4)	52 (66.7)	.905
Pack-years in smokers, median (IQR)	10 (6.8-20)	6 (0-15)	6.5 (0-18)	.828
BMI, mean (SD), kg/m^2	29.9 (6.2)	29.9 (4.7)	29.9 (6.6)	.986
Underweight: BMI <18.5	0			
Normal weight: BMI ≤24.9, n (%)	25 (24)	4 (15.4)	21 (26.9)	.296
Overweight: BMI ≥25, n (%)	33 (31.7)	9 (34.6)	24 (30.8)	.903
Obesity: BMI ≥30, n (%)	46 (44.2)	13 (50)	33 (42.3)	.648
Hurley stage, n (%)				
I	48 (46.2)	9 (34.6)	39 (50)	.256
II	47 (45.2)	13 (50)	34 (43.6)	.733
III	9 (8.7)	4 (15.4)	5 (6.4)	.223
mHSS, median (IQR)	25.5 (13-49)	29 (8-79)	26.5 (13-43)	.671
Comorbidities, n (%)				
Arterial hypertension	13 (12.5)	5 (19.2)	8 (10.3)	.303
Hypothyreosis	11 (10.6)	1 (3.8)	10 (12.8)	.234
Psychiatric disorder	8 (7.7)	3 (11.5)	5 (6.4)	.409
Diabetes mellitus	5 (4.8)	0 (0)	5 (6.4)	.328
Psoriasis	5 (4.8)	2 (7.7)	3 (3.8)	.597
Crohn's disease	2 (1.9)	1 (3.8)	1 (1.3)	.439
Polycystic ovary syndrome	2 (1.9)	—	2 (2.6)	—
Atopic dermatitis	2 (1.9)	0 (0)	2 (2.6)	1

BMI, Body mass index; *HS*, hidradenitis suppurativa; *IQR*, interquartile range; *mHSS*, modified Hidradenitis Suppurativa Score.

effect size is between small (0.10) and medium (0.30) as defined by Cohen.[12]

Differences among groups (n > 2) were examined using the Kruskal-Wallis test, including the Conover post hoc test for pairwise comparisons. A multivariate analysis using logistic regression was performed. Variables with *P* less than .05 were retained in the final model. Associations were summarized using the estimated odds ratio (OR) and the corresponding 95% confidence interval (CI).

Data were analyzed using software (MedCalc, Version 15.2, MedCalc, Mariakerke, Belgium) and *P* less than .05 was considered significant.

RESULTS

In total, 104 patients with HS (26 [25%] men and 78 [75%] women) with a mean ± SD age of 37.7 ± 11.1 (range 18-67) years were included in the study (Table I). Patient characteristics are shown in Table I.

Comparing male and female patients, significantly more female patients with HS had a first-degree relative with HS (*P* = .022) (Table I). The distribution of the affected regions is described in Table II.

Table II. Distribution of affected skin regions in study population

n (%)	Total (n = 104)
Axilla	56 (53.8)
Groin	70 (67.3)
Gluteal	10 (9.6)
Chest	23 (22.1)
Abdomen	9 (8.7)
Genital	17 (16.3)
Perianal	11 (10.6)
Pubis	11 (10.6)

Possible factors influencing disease severity according to the mHSS

MHSS was not significantly different between men and women (*P* = .672) (Table I). There was no correlation between age at onset (*r* = −0.064, *P* = .512) or disease duration (*r* = 0.056, *P* = .571) and mHSS. No significant differences in the mHSS (median [IQR]) between patients with first-degree relatives (30.5 [13.3-47]) and those without first-degree relatives (27 [19-34]) (*P* = .898) or

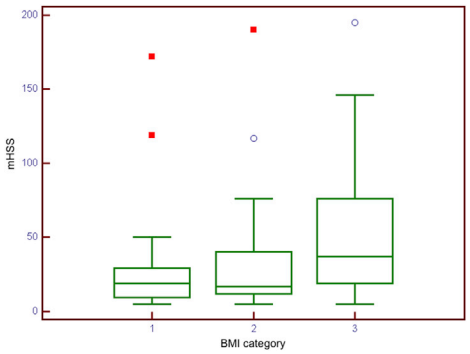

Fig 1. Modified Hidradenitis Suppurativa Score (*mHSS*) versus body mass index (*BMI*) category. There was a significant difference between the normal-weight (*1*) and the obese (*3*) group, and between the overweight (*2*) and obese (*3*) group ($P = .012$, Kruskal-Wallis test). However, not for the normal-weight (*1*) group compared with the overweight (*2*) group.

between nonsmokers (22 [10.3-38.8]) and smokers (32 [13-63.5]) ($P = .205$) were observed.

There was a significant positive correlation between body mass index (BMI) and mHSS ($r = 0.357$, $P = .0002$). Normal-weight patients had a median (IQR) mHSS of 19 (9.5-29.3), overweight had a median (IQR) mHSS of 17 (11.8-40.5), and obese patients had a median (IQR) mHSS of 37 (19-76). Differences among the 3 BMI groups were significant ($P = .012$), except for the normal-weight compared with the overweight group (Fig 1).

Inflammatory serum markers

Data on the CRP levels and WBC were available for all 104 patients. In 98 patients, differential counts were also available. Elevated CRP levels were present in 55 of 104 (52.9%) patients and leukocytosis in 54 of 104 (51.9%). Analysis of the differential counts showed that only the proportion of neutrophils was significantly increased in patients with leukocytosis compared to patients with normal WBC (Table III). Thus, WBC elevation was mainly a result of elevated neutrophil count and in further analysis focus was set on the neutrophil count.

Comparing male and female patients, nonsmoker and smokers, and patients with or without a first-degree relative with HS, there were no significant differences regarding CRP levels (Table IV). Regarding neutrophil count, there was a significantly higher count in smokers ($P = .004$) (Table IV).

There was no correlation between the laboratory parameters and age at onset of the disease or duration of HS (Table V).

Regarding BMI, we found a significant positive correlation only for CRP levels ($r = 0.366$, $P = .0001$). Data showed a significant difference in CRP levels between normal-weight and overweight patients and between normal-weight and obese patients ($P = .002$). There was no difference between overweight and obese patients (Fig 2). Neutrophil counts ($r = 0.039$, $P = .703$) were not correlated with BMI.

Inflammatory serum markers and disease severity

Median CRP level was significantly different among the 3 Hurley groups and increased with the degree of severity. Median (IQR) CRP level for patients classified as Hurley stage I was 2.3 (1.4-6.8) mg/L, 8.1 (3.7-16.1) mg/L for patients classified as Hurley stage II, and 32.5 (18.4-48.6) mg/L for patients classified as Hurley stage III ($P < .0001$) (Fig 3). Median neutrophil count was also significantly different among the 3 Hurley groups, with a median (IQR) neutrophil count of 5.2 (4.4-6) $\times 10^3/\mu$L for patients classified as Hurley stage I, 6.3 (5.3-7.5) $\times 10^3/\mu$L for patients classified as Hurley II, and 7.8 (6.7-9.8) $\times 10^3/\mu$L for patients classified as Hurley stage III ($P = .0002$) (Fig 3).

Regarding disease severity based on mHSS, correlation analysis showed that CRP levels and neutrophil count increased significantly with disease severity. There was a significant positive correlation between CRP level ($r = 0.496$, $P < .0001$) and neutrophil count ($r = 0.330$, $P = .0009$) with mHSS (Fig 4).

Logistic regression model revealed that CRP level was a significant independent predictor for severe disease, defined as Hurley stage III (OR 1.077, 95% CI 1.013-1.145, $P = .016$) (Table VI). Regarding disease severity according to the mHSS, CRP and BMI were significant independent predictors for severe disease, defined as mHSS ≥70 (OR 1.065, 95% CI 1.015-1.117, $P = .009$; OR 1.12, 95% CI 1.009-1.243, $P = .032$, respectively) (Table VII).

DISCUSSION

Our data regarding sex distribution, mean age at onset of HS, and median time between the first symptoms and HS diagnosis correlate well with the literature.[3,4,13-15] The median mHSS of the study population was 25.5. Most patients were classified as Hurley stage I and II and less than 9% as Hurley stage III. This distribution may reflect that we included patients from our outpatient department, which may comprise a group with lower disease severity.

Increased CRP levels were present in 52.9% of the patients, 51.9% had leukocytosis and 25.5% had neutrophilia. Matusiak et al[8,10] reported in 2 studies

Table III. Median percentages of each type of leukocyte in the total number of white blood cells in patients without versus with leukocytosis

Laboratory parameters	Normal range, $\times 10^3/\mu L$	Leukocytosis, % median (IQR)		*P* value
		No	Yes	
Neutrophils	1.8-7.2	58.4 (53.4-63.1)	64.6 (59-68.1)	.001
Lymphocytes	1-4.1	31.9 (26.4-36.1)	25.6 (23-33.4)	.008
Monocytes	0.08-0.8	7.3 (6.2-8.8)	6.2 (5.2-7.9)	.016
Eosinophils	0.04-0.36	1.8 (1.1-2.4)	1.7 (1.2-2.5)	.691
Basophils	<0.08	0.3 (0.2-0.4)	0.3 (0.2-0.4)	.457

IQR, Interquartile range.

Table IV. Median values of C-reactive protein and neutrophils in study population and in each patient group

Characteristic	CRP			Neutrophils		
	n	Median (IQR), mg/L	*P* value	n	Median (IQR), $\times 10^3/\mu L$	*P* value
Total	104	5.9 (1.9-12.1)		98	5.9 (4.7-7.4)	
Male	26	4.3 (1.6-22.8)	.778	26	6.1 (4.3-7.6)	.525
Female	78	6.1 (2-10.5)		72	5.8 (4.7-7.0)	
Nonsmoker	35	0 (0-8.3)	.131	35	4.9 (4-6.6)	.004
Smoker	69	6.5 (0-13.8)		63	6.1 (5.2-7.5)	
No family history (1st degree relative)	84	5.7 (1.9-13.1)	.572	80	5.9 (4.6-7.5)	.653
Family history (1st degree relative)	20	6.2 (1.7-10.5)		18	5.7 (4.9-6.6)	

CRP, C-reactive protein; *IQR*, interquartile range.

Table V. Correlation analysis of laboratory parameters and modified Hidradenitis Suppurativa Score, duration of hidradenitis suppurativa, and age at onset of disease

Laboratory parameters	N	mHSS		Age of onset		Duration of HS	
		Spearman *r*	*P* value	Spearman *r*	*P* value	Spearman *r*	*P* value
CRP	104	0.496	<.001	0.017	.868	0.180	.068
Neutrophils	98	0.305	.002	−0.077	.453	0.085	.408

CRP, C-reactive protein; *HS*, hidradenitis suppurativa; *mHSS*, modified Hidradenitis Suppurativa Score.

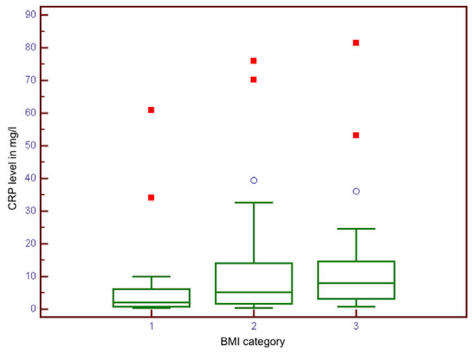

Fig 2. Serum level of CRP vs body mass index (*BMI*) category. There was a significant difference between normal- (*1*) and overweight (*2*) patients, and between normal-weight (*1*) and obese (*3*) patients (P = .002, Kruskal-Wallis test). There was no difference between the overweight (*2*) group compared with the obese (*3*) group.

similar results regarding the proportion of patients with increased CRP levels. In contrast, they reported lower proportions of patients with leukocytosis. However, comparing serum levels of laboratory parameters with available studies is limited because of the heterogeneity of the included patient cohorts, laboratory detection techniques, and the normal range of values.

There was a significant correlation between CRP level and neutrophil count with disease severity according to mHSS. Patients with HS and increased disease severity exhibited elevated CRP levels and neutrophil counts. Furthermore, patients classified in higher Hurley stages had significantly increased serum levels of CRP and neutrophil counts.

CRP is a hepatocyte-derived acute phase protein, produced in the liver in response to IL-6 and has been shown to be a marker of systemic inflammation, elevated in response of inflammatory stimuli. CRP level is subject to natural variations and is, eg, higher in African Americans and in women. This must be considered when interpreting our

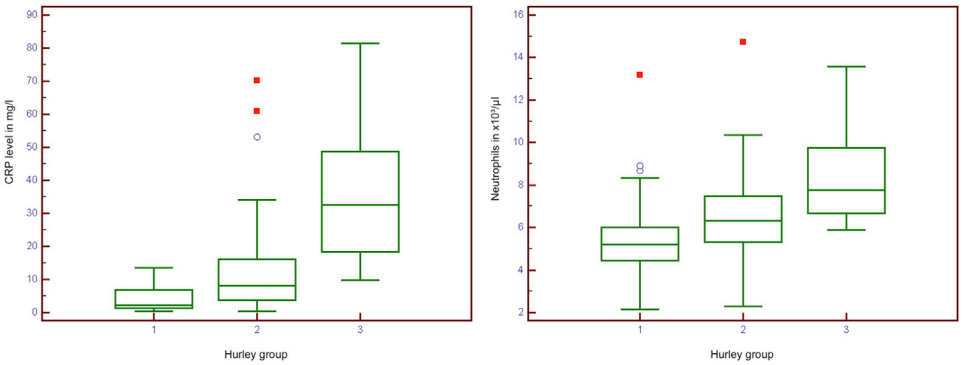

Fig 3. Laboratory parameters (C-reactive protein [*CRP*] and neutrophil count) versus the 3 Hurley groups. The serum levels of CRP ($P < .0001$) and neutrophil count ($P = .0002$) were significantly different between each of the 3 groups and significantly increased with degree of severity (Kruskal-Wallis test).

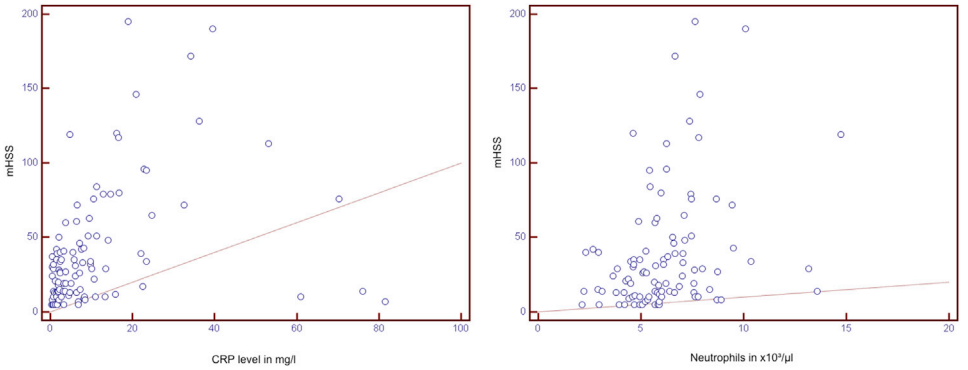

Fig 4. Correlation analysis between laboratory parameters C-reactive protein (*CRP*) ($r = 0.496$, $P < .0001$) and neutrophil count ($r = 0.330$, $P = .0009$) with modified Hidradenitis Suppurativa Score (*mHSS*). Positive correlation according to Spearman.

Table VI. Logistic regression of considered associated factors of disease severity according to Hurley staging system

	Severe vs moderate and mild disease		Moderate and severe vs mild disease	
Variables	**OR (95% CI)**	***P* value**	**OR (95% CI)**	***P* value**
Age at onset, y	1.039 (0.918-1.178)	.538	1 (0.948-1.056)	.976
Disease duration, y	1.003 (0.878-1.146)	.956	1.021 (0.96-1.087)	.497
First-degree relative with HS	0	.995	1.466 (0.418-5.139)	.549
Current smoker	0.185 (0.014-2.382)	.195	0.55 (0.194-1.555)	.260
BMI, kg/m^2	1.15 (0.962-1.374)	.122	0.977 (0.894-1.069)	.623
CRP, mg/L	1.077 (1.013-1.145)	.016	1.205 (1.072-1.354)	.002
Neutrophils, $\times 10^3/\mu$L	1.633 (0.984-2.71)	.057	1.133 (0.866-1.483)	.360

Severe disease (defined as Hurley stage III) and moderate to severe disease (defined as Hurley stage II and III) are the dependent variables.
BMI, Body mass index; *CI*, confidence interval; *CRP*, C-reactive protein; *HS*, hidradenitis suppurativa; *OR*, odds ratio.

data. However, none of our patients was African American. And our analysis showed that both CRP level and neutrophil count were independent of gender, positive family history of HS, age at onset, and duration of disease. The latter indicates that chronic inflammation in HS does not influence these serum markers.

CRP can further be elevated in patients with comorbidities such as arterial hypertension, Crohn's disease, or psoriasis, which occurred in 12.5%, 1.9%, and 4.8% of the included patients.[16-18] However, the influence on CRP level by these factors is minimized because of the facts that none of the patients included had a coronary event reported on

Table VII. Logistic regression of the considered associated factors of disease severity according to the modified Hidradenitis Suppurativa Score

	Severe vs moderate and mild disease		Moderate and severe vs mild disease	
Variables	**OR (95% CI)**	***P* value**	**OR (95% CI)**	***P* value**
Age at onset, y	0.954 (0.879-1.036)	.270	0.94 (0.878-1.007)	.081
Disease duration, y	1.021 (0.942-1.107)	.602	0.98 (0.916-1.049)	.564
First-degree relative with HS	1.034 (0.164-6.496)	.971	3.352 (0.818-13.731)	.092
Current smoker	2.677 (0.494-14.501)	.253	1.485 (0.408-5.403)	.548
BMI, kg/m^2	1.12 (1.009-1.243)	.032	1.205 (1.086-1.336)	<.001
CRP, mg/L	1.065 (1.015-1.117)	.009	1.047 (1.007-1.089)	.018
Neutrophils, $\times 10^3/\mu L$	1.241 (0.904-1.703)	.181	1.285 (0.964-1.712)	.086

Severe disease defined as mHSS ≥70 and moderate to severe disease defined as mHSS ≥40 are the dependent variables.
BMI, Body mass index; *CI*, confidence interval; *CRP*, C-reactive protein; *HS*, hidradenitis suppurativa; *mHSS*, modified Hidradenitis Suppurativa Score; *OR*, odds ratio.

consultation. And because we excluded patients who received antibiotics or immunosuppressive agents within 4 weeks before consultation, none of our patients had active disease. Nonetheless, it should be noted that elevation in CRP levels was identified in otherwise asymptomatic patients with "silent" Crohn's disease.[19]

In accordance to data in literature, the majority (66.3%) of the patients were current smokers.[4,6,20] Smoking was not associated with an increased mHSS, but was significantly associated with increased neutrophil counts. Our findings are in accordance with available data, which showed that leukocytosis and neutrophilia are immune alternations observed in the systemic circulation in smokers.[21-23] CRP levels were not significantly different between nonsmokers and smokers.

Consistent with previous studies, median BMI in this study population was 29.9, and almost 76% of the patients were either overweight or obese.[6,24] Correlation analysis revealed that patients with a higher BMI had a significantly increased disease severity. Studies regarding cardiovascular disease and metabolic syndrome showed that obesity is associated with elevated levels of CRP.[25] Our data showed also a significant positive correlation between increased BMI and elevated CRP levels, whereas there was no correlation between BMI and neutrophil counts.

Multivariate analysis revealed that CRP and BMI were significantly associated with severe disease according to mHSS. Regarding disease severity, according to the Hurley staging system, only CRP was significantly associated with severe disease.

Regarding the role of the inflammatory serum markers for monitoring the response to a systemic anti-inflammatory therapy, Delage et al,[26] who studied 7 patients with HS receiving infliximab, found no significant changes in CRP levels or neutrophil counts before and after treatment. However, other larger trials found a significant reduction of CRP levels after an anti-inflammatory biological treatment.[24,27-29] Based on their findings, Grant et al[27] suggested that CRP may be useful for patient stratification during randomization. In addition to our findings, results from latter studies incorporating higher numbers of patients indicate that especially CRP levels may be useful for monitoring responses to anti-inflammatory therapies. Another clinical implication could be the assessment of subclinical pathogenic alterations in healthy-appearing perilesional skin before clinical onset of HS lesions, namely psoriasiform hyperplasia, follicular plugging, and leukocytic infiltration with increased levels of IL-10, IL-1ß, and tumor necrosis factor-alfa,[30,31] which are not covered by the Hurley staging system nor by the mHSS, but may indeed influence CRP level and neutrophil count. Further, these inflammatory serum markers may provide support in the decision-making process in cases, where it is difficult to classify the clinical findings into a certain Hurley stage, but that rather are pending between Hurley stage I and II or II and III. Based on our data, it is difficult to say whether serum levels of these inflammatory markers may even predict treatment outcome and prognosis of patients with HS. However, prospective studies with healthy controls are necessary to further assess the value of CRP level and neutrophil count for the assessment of disease severity in predicting response to treatment and to analyze the cost-benefit value.

In conclusion, CRP level and neutrophil count are effective tools for assessing the extent of disease severity and grade of inflammation in patients with HS. There was a significant correlation between these inflammatory serum markers and disease severity according to mHSS. With CRP being a

significant and independent predictor for severe disease activity. These markers, especially CRP, which can be easily obtained in routine clinical examinations, should be considered as a valuable extension to the currently available clinical scoring systems for HS.

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