ORIGINAL ARTICLE

Low and high body mass index in hidradenitis suppurativa patients—different subtypes?

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Abstract

Introduction Overweight is a well-established risk factor for hidradenitis suppurativa (HS). In this cross-sectional study, we compare HS patients with a high body mass index (BMI) with HS patients with a low BMI to investigate differences in disease characteristics.

Materials and method Patients were recruited from 17 dermatological centres from four continents. A total of 246 patients with a BMI below 25 were compared to 205 patients with a BMI of above 35.

Results Patients with a high BMI suffered more severe disease (Hurley, physician global assessment, number of areas affected and patient-reported severity (PRS), $P < 0.001$ for all). There was no difference in smoking ($P = 0.783$) nor in family history ($P = 0.088$). In both low and high BMI patients, early onset of HS was a predictor of positive family history ($P < 0.001$, for each). For low BMI patients, an increase in BMI significantly increased PRS ($P < 0.001$). For patients with a high BMI, number of pack-years significantly increased PRS ($P = 0.001$). Cluster analysis of eruption patterns was location specific for low BMI patients but severity specific for high BMI patients.

Discussion Patients with a low and high BMI could represent two clinically different subtypes. We suggest a non-linear relationship between BMI and impact of HS. As patients go from a low BMI patient to a high BMI patient (or from high to low), eruption patterns and risk factors may change.

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Conflicts of interest

Iltefat Hamzavi: Current president of the HS foundation, Investigator for Abbvie and served on an advisory board for Abbvie.

Gregor B.E. Jemec: Honoraria from AbbVie, Inflarx, Leo pharma, Pierre-Fabre and Novartis for participation on advisory boards. Grants from Abbvie, Novartis, Regeneron and Leo Pharma for participation as an investigator. Research grants from Abbvie, Leo Pharma and Novartis.
Ditte Marie Saunte was paid as a consultant for advisory board meeting by AbbVie and received speakers' honoraria and/or received grants from the following companies: Bayer, Abbvie, Desitin, Pfizer, Galderma, Astellas, Novartis and Leo Pharma.
Christos C. Zouboulis: Relevant honoraria from AbbVie, Celgene, Inflarx, Novartis and UCB for participation on advisory boards, as a consultant, and speaker. His department received relevant grants from AbbVie, Biogen, Celgene and Novartis for participation as an investigator.
Other authors stated no conflict of interest.

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Introduction
Hidradenitis suppurativa/acne inversa (HS) is a clinically defined inflammatory skin disease.[1,2] The hallmark of the disease is the recurrence of painful inflamed nodules in the body folds. Nodules become inflamed and progress to abscesses.[3] Progressive lesions such as tunnels (sinus tracts) and secondary lesions, such as scars, develop as the abscesses heal.[1] Untreated, the disease causes obvious and significant morbidity in patients.

In population-based studies, prevalence has been estimated at up to 4% depending on study population.[4] Prevalence estimates from the USA based on insurance database data suggest a lower prevalence of <0.1%.[5,6] Misclassification and/or lack of recognition have been suggested as explanations.[7] Population-based studies in France and Denmark (using questionnaires) have found the prevalence rate to be 1%,[8,9] and recent investigations using validated questionnaire suggest a prevalence of 2.2% (including mild cases) in the general population.[10]

Several observations indicate that the disease is associated with obesity: prevalence and severity are increased in obese populations[11,12]; long-term remission rate is reduced[13]; recurrence of HS in CO_2 laser-treated patients is increased[14]; and finally individual case reports support this association.[15–17] Taken together, these observations suggest that the body mass index (BMI) may constitute a biologically relevant parameter for subgrouping HS patients. As obesity is a modifiable risk factor, the study of obesity in HS is especially important. The purpose of the study was, therefore, to study the differences between obese and non-obese HS patients with the aim of better understanding of the heterogeneity of these patient groups.

Materials and method
An explorative, cross-sectional, descriptive study was conducted based on case note review/interviews and clinical assessment of time-based samples of patients undergoing secondary level care (specialized clinics or hospitals) for HS in a geographically broad sample. All patients above 18 years of age were consecutively screened for inclusion. HS patients with BMI >35 or BMI <25 were eligible for the study. Patients were divided into a low BMI group defined as BMI <25 (underweight and normal weight) and a high BMI group as BMI >35 (obesity class ≥2). To achieve a balanced study population, each centre included patients for the entire study period (1st Nov 2015–1st Sep 2016) or until they had included 20 patients in each category.

Patients were recruited from different outpatient dermatology clinics co-operating with the faster and better research initiative. Nine centres from Europe, two centres each from Africa, the Middle East and North America, and a single centre from Asia participated.

Patients received the questionnaire to be filled out in concert with their dermatologist. The survey explored height, weight, race, smoking habits including pack-years (mean cigarettes per day multiplied with the number of years smoked divided by 20), daily/weekly alcohol consumption, family history of HS, disease impact as measured by PRS (numeric rating scale 0–10), flares during the past 6 months for the regions: axilla, under or between breasts, buttocks, groin, pubic area, genitals, anal area and elsewhere (ordinal scale: none, 1–2, 3–10 or more than 10). Hurley score, Physician Global Assessment (PGA, 0–5, 0 = no activity, 5 = more than 5 abscesses or draining fistula) and comorbidities such as acne, joint problems, gastrointestinal problems and kidney disease.

Statistics
Numeric data are presented with mean and standard deviation or median and interquartile range depending on normality, and differences between groups were examined using a *t*-test or a Mann–Whitney *U*-test as appropriate. Categorical data are presented with frequency and percentages, and comparisons between groups were done with a chi-squared test or Fishers exact test for nominal data and with Kruskal–Wallis test for ordinal data. Multiple linear and logistic regressions were

performed using the stepwise, forward and backward approach, using estimate changes to assess confounding. Akaike's information criterion was used to assess the regression models. Ordinal regression models were performed, including test for parallel lines. Two-step cluster analysis was performed to classify patients based on outbreaks the past 6 months assigning ranks 0 to 3, to outbreak frequency of 0, 1–2, 3–10 and more than 10. All statistics were performed in R 3.31 (GNU, General Public License).

Results

A total of 491 participated in the survey: 246 patients with low BMI and 205 with a high BMI were included. Forty patients were excluded, as they did not meet the inclusion criteria of BMI over 35 or below 25. Patient demographics and risk factors including *P*-value for differences between the groups are presented in Table 1. Patient clinical characteristics and comorbidities as well as *P*-value for differences between the groups are shown in Table 2.

Patients with a high BMI suffered a more severe disease than patients with low BMI, both objectively based on highest Hurley score described ($P < 0.001$), PGA score ($P < 0.001$) and number of areas affected ($P < 0.001$) and subjectively based on PRS ($P < 0.001$). In ordinal regression models, patients in the high BMI group had an odds ratio of 1.99 (CI 95%: 1.4–2.80) of having a higher PGA score than low BMI patients, and an odds ratio of 2.61 (CI 95%: 1.86–3.66) of having more areas affected. Other risk factors (smoking, sex, family history) were eliminated in the models as non-significant. Odd ratio for Hurley staging could not be assessed due to violation of the parallel lines test for Hurley staging.

Obese patients were significantly older than patients with low BMI (median age 33 vs. 38, $P < 0.001$). High BMI patients were also older at age of first symptoms (median 20 vs. 23, $P < 0.001$) even after correcting for current age. Obese patients reported more axial and peripheral joint problems ($P = 0.001$ and $P = 0.033$, respectively).

Patients with low BMI reported a significantly higher alcohol consumption ($P = 0.007$) and were more prone to acne ($P = 0.007$), but these differences disappeared after correcting for age. No difference was detected between the groups in familiar disposition of HS ($P = 0.088$) and smoking status ($P = 0.783$).

Table 1 Patient demographics and risk factors

Patient demographics and risk factor	Low BMI *N* = 246	High BMI *N* = 205	*P*-value
Female, *N* (%)	155 (62.0)	126 (61.4)	0.986
Mean age, (SD)	22.3 (1.9)	38.4 (3.9)	0.000*
Median BMI, (IQR)	22.6 (3.1)	37.1 (3.8)	0.000*
White/Caucasian, *N* (%)	209 (83.6)	182 (88.8)	0.151
Other race, *N* (%)	37 (16.4)	23 (11.2)	0.151
Smoker, *N* (%)	130 (52.8)	111 (54.1)	0.783
Alcohol: Never, *N* (%)	133 (54.1)	135 (65.9)	**0.007**†
Alcohol: 1–2 week, *N* (%)	99 (40.2)	65 (31.7)	**0.007**†
Alcohol: Daily, *N* (%)	14 (5.7)	5 (2.4)	**0.007**†
Positive family history, *N* (%)	58 (23.6)	63 (30.7)	0.088
Median age at first boil (IQR)	20 (11)	23 (18)	0.001‡

*Statistically significant.
†Univariably statistically significant but no difference after adjusting for age.
‡Statistically significant difference after correcting for current age.
Grouped *P*-values in bold represent a single Kruskal–Wallis test.
BMI, body mass index, *N*, number; IQR, interquartile range; SD, standard deviation.

Table 2 Patient clinical appearance and comorbidities

Patient clinical appearance	Low BMI *N* = 246	High BMI *N* = 205	*P*-value
PRS, median (IQR)	5 (4)	7 (5)	0.000*
Hurley I, *N* (%)	92 (37.4)	56 (27.3)	**0.000***
Hurley II, *N* (%)	124 (50.4)	87 (42.2)	**0.000***
Hurley III, *N* (%)	30 (12.2)	62 (30.2)	**0.000***
PGA median, (IQR)	2 (1)	3 (3)	0.000*
Number of areas affected, mean (SD)	2.5 (1.6)	3.47 (2.0)	0.000*
Axial joint problems, *N* (%)	13 (5.3)	30 (14.6)	0.001*
Peripheral joint problems, *N* (%)	34 (13.8)	44 (21.5)	0.033*
GI-problems, *N* (%)	24 (9.8)	31 (15.1)	0.112
No facial acne, *N* (%)	168 (68.3)	163 (79.8)	**0.007**†
Mild facial acne, *N* (%)	38 (15.4)	23 (11.2)	**0.007**†
Moderate facial acne, *N* (%)	30 (12.2)	12 (5.9)	**0.007**†
Severe facial acne, *N* (%)	10 (4.1)	7 (3.4)	**0.007**†
Truncal acne, *N* (%)	32 (13.0)	18 (8.8)	0.154
Kidney disease, *N* (%)	1 (0.4)	4 (2.0)	0.182

*Statistically significant.
†Univariably statistically significant but no difference after adjusting for age.
Grouped *P*-values in bold represent a single Kruskal–Wallis test.
IQR, interquartile range; *N*, number; PGA, physician global assessment SD, standard deviation.

Positive family history predictors

A logistic regression was performed examining possible predictors for having a positive family history of HS, to examine common characteristics for patients with a positive family history. The regression was performed in the two groups separately (Table 3). The possible predictors were BMI (numeric), smoking status (yes/no), PRS (numeric), acne severity (ordinal), alcohol consumption (ordinal), age (numeric) and age at diagnosis (numeric) and 1st order interactions between each variable.

For patients with a low BMI backward, forward and stepwise regression models excluded all predictors for positive family history as insignificant and non-confounding, except age and age at diagnosis. Older age at first symptom provided an odds ratio (OR) of 0.72 (0.58–0.87, $P = 0.001$) for positive family history (Note OR <1). Similarly, older patients had an OR of 1.16 (1.01–1.34, $P = 0.04$). The omnibus goodness-of-fit test had a P value of 0.79, suggesting that the model explained more than the null hypothesis.

Table 3 Predictors for positive family history—logistic regression

	Low BMI group				High BMI group			
	OR	2.5%	97.5%	*P*-value	OR	2.5%	97.5%	*P*-value
Per 5 years increase of age	1.16	1.01	1.34	0.04	1.36	1.14	1.64	<0.001
Per 5 years increase of age at diagnosis	0.72	0.58	0.87	0.001	0.67	0.54	0.81	<0.001

Predictors examined were BMI (numeric), smoking status (yes/no), patient-reported severity (numeric), acne severity (ordinal), alcohol consumption (ordinal), age (numeric) and age at diagnosis (numeric) and 1st order interactions between each variable, and the table shows the odds ratio (95% CI) and significance level for the predictors not excluded by the model. The regression was performed for the low and high BMI group separately but yielded the same variable as predictors in the final model.
BMI, body mass index; OR, odds ratio; PRS, patient-reported severity.

For patients with a high BMI, the same significant predictors were found, age and age at diagnosis. Older age at first symptom provided an OR of 0.67 (0.54–0.81, $P = 0.001$) for positive family history (Note OR <1), and older patients had an OR of 1.36 (1.14–1.64, $P < 0.001$). The omnibus goodness-of-fit test had a P value of 0.23, again suggesting that the model explained more than the null hypothesis.

Patient-reported severity predictors

Linear regression was performed to identify predictors for the PRS score. Considered predictors were as follows: sex (dichotomous), family history (dichotomous), BMI (numeric), pack-years (numeric), age (numeric), age at diagnosis (numeric) and the interaction between sex and BMI and the interaction between pack-years and sex.

In patients with low BMI, the final model included sex and BMI (Table 4). For this model, a single outlying observation was omitted, as the residual vs. leverage plot for the model suggested an undue influence of this data point on the overall model.

Low BMI patients

An increase in BMI significantly increased PRS (0.37; $P < 0.001$). Female sex was associated with a trend towards more severe disease in this group (0.627, $P = 0.07$).

Table 4 Changes in patient-reported severity (PRS) (0–10)—linear regression

Low BMI group	Estimate	*P*-value
Sex (female)	0.627	0.07
Per 1 increase in BMI	0.374	<0.001
High BMI group		
Per 1 increase in BMI	0.38	0. 185
Pack-years	0.08	0.001
BMI x pack-years	−0.01	0.002

Predictors examined were sex (dichotomous), family history (dichotomous), BMI (numeric), pack-years (numeric), age (numeric), age at diagnosis (numeric) and the interaction between sex and BMI and the interaction between pack-years and sex. The table shows the coefficient values and the significance level for the included predictors. The regression was performed for the low and high BMI group separately.
BMI, body mass index.

High BMI patients

The same model was examined including the above-mentioned predictors after the exclusion of two outliers determined by the residuals vs. leverage plot. In this group, pack-years appeared associated with increased PRS (0.38, $P = 0.001$) but not BMI. The interaction between BMI and pack-years is slightly negative suggesting a ceiling effect or an additive but not synergistic effect of the parameters.

Eruption pattern clustering

Two-step cluster analysis of patients with a low BMI divided patients into three clusters, which we then labelled, based on the frequency distribution: the infrequent eruptions group (52.4%), the pubic/genital group (25.6%) and the axillary/mammae group (22.0%). The infrequent eruptions group was characterized by a medium frequency in the axilla and a comparatively lower frequencies in all other anatomical areas. The pubic/genital group was characterized by a comparatively high frequency in the pubic and genital region but the lowest frequency of the axillary and no eruptions in the mammae region.

Table 5 Mean frequency rank and mean PRS in each location for clusters of low body mass index patients

Cluster low BMI	Infrequent eruptions (*N* = 129)	Pubic/genital (*N* = 63)	Axillary/breasts (*N* = 54)
Axillary	0.71	0.44	1.44
Mammae	0.03	0.00	0.56
Buttocks/Nates	0.29	0.51	1.56
Groin	0.63	1.32	1.37
Pubic area	0.09	1.16	0.33
Genitals	0.10	1.19	0.37
Anal area	0.05	0.52	0.54
Elsewhere	0.02	0.03	0.72
Evaluation variable (not used for the clustering)			
Mean PRS	4.54	6.35	7.11
Mean BMI	22.22*	22.33*	22.68*
Female, *n* (%)	77 (60)†	42 (67)†	32 (60)†

*No statistical difference in a Kruskal–Wallis test ($P = 0.277$).
†No statistical difference in a chi-squared test ($P = 0.662$).
BMI, body mass index; PRS, patient-reported severity.

Table 6 Mean frequency rank and mean PRS in each location for clusters of high body mass index patients

Cluster high BMI	Mild (*N* = 98)	Moderate (*N* = 81)	Severe (*N* = 26)
Axillary	1.06	1.20	2.35
Mammae	0.19	0.45	2.04
Buttocks/Nates	0.12	0.92	1.96
Groin	0.77	1.24	2.58
Pubic area	0.12	1.02	2.15
Genitals	0.05	0.62	1.65
Anal area	0.02	0.39	1.00
Elsewhere	0.10	0.58	0.81
Evaluation variable (not used for the clustering)			
Mean PRS	5.33	7.02	8.00
Mean BMI	37.82*	38.36*	40.27*
Female, *n* (%)	57 (58)†	49 (60)†	20 (77)†

*No statistical difference in a Kruskal–Wallis test ($P = 0.091$).
†No statistical difference in a chi-squared test ($P = 0.212$).
BMI, body mass index; PRS, patient-reported severity.

The axillary/mammae group had the highest frequency in the axillary, breast and buttocks area. There was no difference in BMI ($P = 0.277$) or gender ($P = 0.662$) between the groups (Table 5).

For *patients with* a *high BMI,* the clusters were likewise divided into three groups, we labelled these: the mild (47.8%), the moderate (39.5%) and the severe group (12.7%). The mild group had the lowest frequency in all areas, the moderate had the medium frequency in all areas and the severe group had the highest. There was no difference in BMI ($P = 0.091$) or gender ($P = 0.212$) between the clusters (Table 6.)

Discussion

Several important differences were found between obese and normal weight HS patients suggesting that BMI may be a relevant factor for subclassification. We found that obese patients had more severe disease, had different risk factors for disease impact and different eruption patterns.

Obesity as a risk factor for HS occurrence and severity is supported by retrospective, cross-sectional and prospective observations (9–12). In agreement with the literature, we found that high BMI HS patients have more severe disease than low BMI HS patients. We also found that high BMI patients reported more joint problems. Joint problems in HS have previously been studied by Richette *et al.*[18] who found the majority of joint problems may be explained as obesity-related.[19]

Genetic disposition was found associated with an earlier age of onset and older current age. We suggest that patients who report a positive family history are older, simply because member of their family required longer time to develop HS. This indicates that some patients without a positive family history may develop one later in life and that results based on positive family history should therefore be interpreted with caution. Deckers et al. (2015) have found an association between widespread disease, early onset and a positive family history.[20] In the present study, we found more areas affected in patients with positive family history in the high BMI patients group ($P < 0.001$) but not in the low BMI patients ($P = 0.604$). This may indicate a synergistic effect between BMI and a genetic susceptibility for widespread disease. Other measurements of severity such as PRS, Hurley stage and PGA did not differ between the groups. (Table S1).

The Sartorius scoring system[12] was not included in this study as it is not used routinely by all participating centres. We, therefore, have focused on PRS as a surrogate measure of disease impact and severity. Surprisingly, disease impact was significantly affected by BMI in the low BMI patient group and did only show a trend towards significance in the obese patients. This may reflect that the PRS is influenced in general by BMI but that a ceiling effect exists, that is that once a threshold is reached subsequent increases of BMI matters less to PRS. Threshold effects for BMI in HS patients have previously been suggested.[21]

Smoking as a risk factor is less well established than BMI.[5,9,12,13,22] The results of this study suggest that the pack-years exert a synergistic effect with obesity. The cumulative number of cigarettes smoked influences PRS but only after crossing a threshold BMI. Estimating the threshold is impossible without the full range of BMI's and thus beyond the scope of this paper. Being a current smoker did not influence the regression model, indicating that pack-years are more important than whether you are a current smoker or not.

For patients with a low BMI, cluster analysis divided them into three groups: a large infrequent eruptions group (52.4%) and two smaller pattern specific groups, the pubic/genital group (25.6%) and the axillary/mammary group (22.0%). It is noteworthy that the same subgrouping is not found in the high BMI group. It is likely that for the high BMI patients (BMI 35+), a high frequency of eruptions in one area is correlated with simultaneous eruptions in all other areas, that is more generalized disease flares that may be interpreted as systemic. Interestingly, we found only a trends towards difference ($P = 0.091$) in BMI between the generated clusters (mild, moderate and severe) for high BMI patients, and this further supports our findings that after a threshold BMI is crossed, BMI seems to matters less for severity. The clustering was not an attempt to create phenotypes of HS patients, which we believe requires the addition of morphology, but it was merely an attempt to elucidate and report if any eruptional patterns were evident. It may be speculated that any flare pattern you have as a low BMI patient slowly erodes as BMI increases.

The data collected for this study suggest a number of differences between the high BMI and the low BMI HS patients. Obesity class II (35.0–39.99) and III (≥40.0) patients suffer from more severe, widespread disease causing greater impact. BMI is

the most important risk factor for impact of the disease in the low BMI group, while number of pack-years is most important in high BMI patients. Additionally, patterns of flares are only evident in patients with a low BMI. Combined with the knowledge that patients with a high BMI respond less well to treatment, it appears attractive to suggest that patients with low and high BMI form two clinically different HS subtypes.

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Supporting information

Additional Supporting Information may be found in the online version of this article:

Table S1. Other severity measures for low and high Body Mass Index patients.