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Hidradenitis suppurativa in Black and White patients – a clinical study

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Abstract. – OBJECTIVE: It is suggested that hidradenitis suppurativa (HS) is more prevalent and causes greater morbidity in Black patients than in White. Clinical data are however lacking.

PATIENTS AND METHODS: We therefore describe HS risk factors, disease severity and clinical phenotypes in the Blacks and Whites. Patients referred for HS between 1984 and 2019 at the Johns Hopkins Hospital were identified using the Pathology Data System (PDS). Clinical and sociodemographic characteristics were extracted and the van der Zee & Jemec HS clinical phenotypes were recovered.

RESULTS: A total of 278 patients were identified. Ethnically, 108 (38.8%) were White, and 170 (61.2%) Black. The following HS phenotypes were found: Regular (n=193, 69.4%), scarring folliculitis (n=40, 1.4%), frictional furuncle (11.2%), conglobata (n=9, 3.2%), syndromic (n=3, 1.1%) and ectopic (n=2, 0.7%). No statistically significant ethnic differences in clinical presentation were found. Blacks however had more severe diseases than Whites (p= 0.024 for trend). With multivariate logistic regression analysis, we found that male sex, disease duration, and smoking were independent predictors of regular HS phenotype. Major limitations are the limited number of cases studied and the lack of data regarding response to therapies.

CONCLUSIONS: Demographics and phenotypical presentation of HS patients do not seem to be associated with Black ethnicity. However, there is a significant trend for Blacks to present with more Hurley stage 2 and 3 disease compared to White patients. It is speculated that ethnic differences are epiphenomena to social factors, highlighting the broader importance of ethnicity.

Key Words:
Hidradenitis suppurativa, Clinical phenotypes, Ethnicity, Blacks, Whites.

Introduction

Ethnicity may play a role in Hidradenitis suppurativa (HS)[1]. The link between ethnicity and HS is hampered by the fact that most published studies[2] predominantly are based on overwhelmingly White populations, e.g., in Vazques et al's population-based survey[3], based in Olmsted

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County, Minnesota (USA) 90.3% of the patients were White reflecting the demography of the sampled region.

A generally higher prevalence rate has however been suggested in US Black population, where the prevalence has been estimated at 0.3% [95% confidence interval (CI): 2.9-3.0] compared to 0.095% (95% CI: 0.094-0.096) in the White population[4]. In contrast, a pilot study[5] of HS prevalence in Africa (Ghana) suggests a prevalence rate of 0.8% (0.2-2.0). Asian patients appear to differ from Whites in sex and age[6,7].

It has additionally been suggested that the HS may not only be more common, but also more florid in Blacks[4]. Studies[4-7] on clinical severity in different ethnicities are however scarce. Given the lack of data, we compared Black and White HS patients with a particular attention for potential differences in risk factors, disease severity and clinical phenotypes.

Patients and Methods

Patient Selection: Inclusion and Exclusion Criteria

This is a single institution retrospective study. The Pathology Data System (PDS) was mined for all patients with a diagnosis of HS between 1984 and 2019. Only Johns Hopkins patients were included. Search results were extracted from medical charts and records belonging to John Hopkins University and included: name, age at diagnosis, age at study production, gender, disease duration, co-morbidities, smoking status, body mass index (BMI), family history of HS, presence of pilonidal cysts, and ethnicity.

Disease severity was assessed using the maximum Hurley score[8], which classifies patients according to the most severely affected areas and depends on the identification of inflamed lesions and permanent lesions (scars and tunnels). Three stages are defined:

Stage 1: Abscess formation, single or multiple, without sinus tracts (tunnels) and scarring.

Stage 2: Recurrent abscesses with tract formation and granulation, single or multiple, widely separated lesions.

Stage 3: Diffuse or near diffuse involvement, or multiple interconnected tracts and abscesses across entire area.

The patient identifiers were removed and replaced with a de-identified study label. This de-identified dataset was shared with the dermatology study team in order to examine HS clinical phenotypes (regular, frictional furuncle, scarring folliculitis, conglobata, syndromic and ectopic)[9], in the context of demographic information. Diagnoses performed before Dessau diagnostic criteria publication in 2015[10] were revalidated with clinical information collected and in the case of inaccuracy it was excluded from analysis.

HS clinical phenotypes were diagnosed according to the following criteria[9]:

- Regular type: patients that fulfilled Dessau criteria and did not meet the specific criteria of the other types listed below.
- Frictional furuncle type: overweight/obese patients with regular type plus abscesses and deep-seated nodules located in high-friction areas, namely waistline, buttocks and thighs. Rarely have fistulas.
- Scarring folliculitis type: overweight/obese patients with regular type with pubic and perineal area involved by several superficial polymorphic lesions (e.g., pustules, cysts, comedones, superficial nodules). Rarely have fistulas and usually Hurley stage 1.
- Conglobata type: patients with regular type plus numerous inflammatory cysts involving back and face (acne conglobate). The prototypic patient is a man with normal BMI and moderate to severe disease (Hurley stage 2 and 3);
- Syndromic type: patients grouping HS with other articular and dermatological manifestations previously reported in the systematic review by Gasparic et al[11];
- Ectopic type: patients with regular type and/or clinical manifestations similar to HS on the face or in positions other than the back, armpits, pubic, perineum, buttocks and thighs.

The Institutional Review Committee approved the study protocol that fulfilled the principles of the Helsinki Declaration of 1975, revised in 2008 (https://www.wma.net/what-we-do/medical-ethics/declaration-of-helsinki/) and patients signed a written consent form.

Statistical Analysis

Prior to any statistical processing, data were visually inspected to capture any potential outliers. Normality of data distribution was assessed with the D'Agostino-Pearson *omnibus* test. Continuous

variables were expressed as mean ± standard deviation, while categorical parameters were computed as percentages, where appropriate.

The Chi-squared test was used to compare categorical variables between two groups, whereas Chi-squared test for trend was conducted for ordinal parameters (such as smoking status or Hurley stage). Student's *t*-test (or its non-parametric version, the Mann-Whitney U test, based on data distribution) was computed for capturing differences between the two ethnic groups. Multivariate multinomial and binomial logistic regression analyses were carried out for shedding light on associations of Hurley stage and HS phenotype (regular *vs.* non regular phenotypes), respectively.

All statistical analyses were conducted with "Statistical Package of Social Sciences" (SPSS for Windows, version 24.0, IBM Corp., Armonk, NY, USA). For all statistical analyses, figures with *p*-values less than or equal to 0.05 were considered statistically significant.

Results

A total of 278 patients were identified: 72 males (25.9%) and 206 females (74.1%), with a female-male ratio of 2.9:1 and a mean age at diagnosis of 37.36±13.47 years and at the time of data review of 51.81±14.23 years. Average disease duration was 14.50±7.35 years. Ethnically, 108 (38.8%) were White whereas 170 (61.2%) where Black (black/white ratio of 1.6:1).

Risk

A minority of Black and White patients, n=58 (20.9%) and n=39 (14.0%) were current and former smokers, respectively. Only four patients (1.4%) had a family history of HS. Additional descriptive statistics are provided in Table I.

Univariate analysis (Table II) indicated that Whites and Blacks differed only for age at diagnosis (39.43±14.20 years *vs.* 36.05±12.85 years, p=0.043), age at data collection (55.16±12.75 years *vs.* 49.73±14.74 years, p=0.002), and disease duration (15.82±6.62 years *vs.* 13.67±7.68 years, p=0.020).

Smoking status achieved a borderline significance (p=0.081, and p=0.075 for trend), with current and former smokers more represented amongst White (n=26, 24.1%, *vs.* n=32, 18.8%, for current smokers; n=20, 18.5%, *vs.* 19, 11.2%, for former smokers).

Multivariate analysis indicated no differences between gender, family history of HS and presence of pilonidal cysts.

Severity

Concerning the severity of the disease, 105 subjects (37.8%) had a maximum Hurley stage 1, whereas 115 (41.4%) and 58 (20.9%) a maximum Hurley stage 2 and 3, respectively. Further details are shown in Table I. Two hundred and forty-one patients (86.7% of the sample) suffered from at least one systemic ICD coded co-morbidity.

Multivariate analysis regarding disease severity indicated a trend (p=0.024 for trend) with Hurley stage 1 more present in Whites (n=50, 46.3%, *vs.* n=55, 32.4%), while stage 2 and 3 was more represented amongst Blacks (n=75, 44.1%, *vs.* n=40, 37.0% for grade 2; n=40, 23.5%, *vs.* n=18, 16.7% for grade 3).

No differences were found between gender, co-morbidities, HS phenotype, family history of HS and presence of pilonidal cysts.

Clinical Type

All phenotypes were represented in the study and there were comparable numbers of patients in all

Table I. Primer sequences.

Parameter	
Age at diagnosis	37.36 ± 13.47; 37 [14-81]
Age at study production	51.81 ± 14.23; 53 [18-90]
Gender	
- Male	72 (25.9%)
- Female	206 (74.1%)
Disease duration	14.50 ± 7.35; 13.5 [1-35]
Co-morbidities	241 (86.7%)
Smoking	
- Current smokers	58 (20.9%)
- Former smokers	39 (14.0%)
- Non-smokers	181 (65.1%)
Phenotype	
- Regular	193 (69.4%)
- Frictional furuncle	31 (11.2%)
- Scarring folliculitis	40 (14.4%)
- Conglobata	9 (3.2%)
- Syndromic	3 (1.1%)
- Ectopic	2 (0.7%)
Hurley	
- Grade 1	105 (37.8%)
- Grade 2	115 (41.4%)
- Grade 3	58 (20.9%)
Family history	4 (1.4%)
Pylonidal cist	29 (10.4%)
Ethnicity	
- White or Caucasian	108 (38.8%)
- Black	170 (61.2%)

Table II. Univariate analysis on the differences between white or Caucasian and black or Black hidradenitis suppurativa (HS) patients.

Parameter	White (n = 108)	Black (n = 170)	Statistical significance
Age at diagnosis	39.43 ± 14.20; 38 [16-81]	36.05 ± 12.85; 35 [14-73]	0.043
Age at data extraction	55.16 ± 12.75; 56 [29-84]	49.73 ± 14.74; 51 [18-90]	0.002
Gender			0.570
- Male	30 (27.8%)	42 (24.7%)	
- Female	78 (72.2%)	128 (75.3%)	
Disease duration	15.82 ± 6.62; 15 [1-35]	13.67 ± 7.68; 12 [2-34]	0.020
Co-morbidities	25 (23.1%)	145 (85.3%)	0.391
Smoking			0.081
- Current smokers	26 (24.1%)	32 (18.8%)	(0.075 for trend)
- Former smokers	20 (18.5%)	19 (11.2%)	
- Non-smokers	62 (57.4%)	119 (70.0%)	
HS phenotype			0.579
- Regular	79 (73.1%)	114 (67.1%)	
- Frictional furuncle	12 (11.1%)	19 (11.2%)	
- Scarring folliculitis	12 (11.1%)	28 (16.5%)	
- Conglobata	4 (3.7%)	5 (2.9%)	
- Syndromic	0 (0.0%)	3 (1.8%)	
- Ectopic	1 (0.9%)	1 (0.6%)	
Hurley			0.058
- Grade 1	50 (46.3%)	55 (32.4%)	(0.024 for trend)
- Grade 2	40 (37.0%)	75 (44.1%)	
- Grade 3	18 (16.7%)	40 (23.5%)	
Family history of HS	2 (1.9%)	2 (1.2%)	0.646
Pylonidal cist	9 (8.3%)	20 (11.8%)	0.363

Hurley stages. The most common HS phenotype was regular (n=193, 69.4%), followed by scarring folliculitis (n=40, 14.4%), frictional furuncle (n=31, 11.2%), conglobata (n=9, 3.2%), syndromic (n=3, 1.1%) and ectopic (n=2, 0.7%). With the multivariate multinomial logistic regression analysis (Table III), disease duration [OR:0.94 (95% CI 0.90-0.98), p=0.004] and smoking status [OR 0.37 (95% CI 0.17-0.80), p=0.011, for non-smoker *vs.* current smoker] were independent predictors of Hurley stage 2, whereas disease duration [OR:0.91 (95%CI 0.86-0.96), p=0.000], and smoking status [non-smoker *vs.* current smoker, OR:0.34 (95%CI 0.14-0.83), p=0.018; former smoker *vs.* current smoker, OR:0.33 [95%CI 0.10-1.17], p=0.086, borderline significant]. With multivariate logistic regression analysis (Table IV), ethnicity failed to achieve statistical significance (p=0.462) as an independent predictor of HS phenotype.

Discussion

It has been suggested that HS is both more prevalent and severe in Blacks than in Whites, but little is known[2,4,12,13]. The majority of data are derived from predominantly White populations. We have therefore compared the risk factors for developing HS, the disease severity and the clinical classification of Black and White HS patients in a mixed population. Overall, our mixed cohort of patients was comparable to previously published cohorts regarding basic demographics, such as age and sex.

Blacks were younger when included in the study and had a slightly shorter disease duration. Only a trend was found for more severe HS in Black patients, with more having Hurley stage 2 or 3 (p=0.024 for trend). Apart from this trend, the differences are likely due to the overall younger age of Blacks. It is therefore speculated that other factors may play a role. In particular socio-economic confounders are suspected to be of importance, e.g., insurance type. Unfortunately, insurance data based on ethnicity were not available[14,15].

Previous studies[16-19] in different populations, i.e., United States, Australia, Denmark and the Netherlands, have suggested that HS is associated with lower socio-economic status, lower income, and increased unemployment in comparison to non-HS populations[20,21]. Socioeconomic status and ethnicity could be regarded as key factors and physicians should be aware of their importance in long-term treatments. In addition to the inherent problems associated with lower social

Table III. Multivariate multinomial logistic regression analysis, investigating co-variates associated with Hurley stage.

Hurley	Parameters	B	S.E.	Wald	Statistical significance	OR	95% CI	
							Lower bound	Upper bound
Grade 2	Intercept	3.07	0.81	14.34	0.000			
	Age at study production	-0.02	0.01	2.16	0.142	0.98	0.96	1.01
	Gender (female -0.17 *versus* male)	0.35	0.22	0.638	0.85	0.43	1.68	
	Disease duration	-0.06	0.02	8.23	0.004	0.94	0.90	0.98
	Ethnicity (white *versus* black)	-0.46	0.30	2.27	0.132	0.63	0.35	1.15
	Smoking (non-smoker *versus* current smoker)	-1.00	0.39	6.47	0.011	0.37	0.17	0.80
	Smoking (former smoker *versus* current smoker)	-0.52	0.51	1.04	0.308	0.60	0.22	1.61
Grade 3	Intercept	2.77	0.94	8.67	0.003			
	Age at study production	-0.01	0.01	0.31	0.580	0.99	0.97	1.02
	Gender (female *versus* male)	-0.54	0.40	1.78	0.182	0.58	0.26	1.29
	Disease duration	-0.10	0.03	12.21	0.000	0.91	0.86	0.96
	Ethnicity (white *versus* black)	-0.58	0.37	2.37	0.124	0.56	0.27	1.17
	Smoking (non-smoker *versus* current smoker)	-1.07	0.45	5.59	0.018	0.34	0.14	0.83
	Smoking (former smoker *versus* current smoker)	-1.10	0.64	2.96	0.086	0.33	0.10	1.17

status, these associations may also all influence patients' ability to self-care and health-seeking behavior[22,23].

In spite of these basic similarities, ethnicity may still influence the phenotype. Various HS subtypes have been defined. Based on latent class analysis of a cohort of 618 patients Canoui-Poitrine et al[24] proposed three subgroups of HS according to location and lesion type: axillary-mammary, follicular, and gluteal. In 2015, more HS phenotypes were suggested by van der Zee and Jemec based on clinical experience[9]. These included: regular type, frictional furuncle type, scarring folliculitis type, conglobate type, syndromic type, and ectopic type[9]. The van der Zee & Jemec phenotypes were chosen as they are independent of severity and each phenotype may include patients in all three Hurley stages, which allows this classification system to be utilized broadly. This study therefore also serves as explorative in a cohort of phenotypes in 278 patients[9].

No significant differences were however found, and ethnicity failed to achieve statistical significance in multivariate logistic regression analysis. This implies that while Black HS patients are younger and possibly have more se-

Table IV. Multivariate logistic regression analysis investigating co-variates associated with hidradenitis suppurativa (HS) phenotype (regular versus non-regular).

Parameters	B	S.E.	Wald	Statistical significance	OR	95% CI	
						Lower	Upper
Age at diagnosis	0.00	0.01	0.00	0.950	1.00	0.98	1.02
Gender (male versus female)	0.70	0.31	5.02	0.025	2.02	1.09	3.74
Disease duration	0.06	0.02	7.46	0.006	1.06	1.02	1.11
Smoking			8.54	0.014			
Smoking (former smoker versus non-smoker)	0.90	0.34	7.27	0.007	2.47	1.28	4.75
Smoker (current smoker versus non-smoker)	0.18	0.44	0.16	0.690	1.19	0.50	2.83
Ethnicity (Black versus White)	0.22	0.230	0.54	0.462	1.24	0.70	2.22
Constant	-1.20	0.71	2.85	0.092	0.30		

vere disease, they do not present with a special clinical presentation; again, underlining the likelihood that other factors play a role in the higher morbidity.

Looking the specific phenotypes, male sex, disease duration, and smoking status were independent predictors of the regular HS phenotype. In fact, males in this study were over twice as likely to have a regular HS phenotype as females. This association suggests that females may be more likely to present a different phenotype of HS. This finding raises many future questions, including if pathogenic processes in females, such as hormone imbalances may subject them to having non-regular forms of HS that are more difficult to treat, leading to increased disease burden in females.

Strengths and Limitations

This study addresses possible differences between Black and White HS patients by using data mining with a reproducible string. Limitations mainly consist of those inherent to the retrospective design, the limited number of cases studied and the lack of data regarding response to therapies.

Conclusions

This study shows that the demographics and phenotypical presentations do not seem to be associated with ethnicity. Black HS patients closely resemble white HS patients. We did however find a trend for Blacks to present with more advanced disease, i.e., having Hurley stages 2 or 3. In the absence of clinical differences it may be speculated that socioeconomic factors play a role (i.e., exposome, methylome or diet[25-28]), highlighting the need for awareness to these factors in the management of HS.

Conflict of Interest

GBE Jemec has received honoraria from AbbVie, Chemocentryx, Coloplast, Incyte, Inflarx, Novartis, Pierre Fabre and UCB for participation on advisory boards, and grants from Abbvie, Astra-Zeneca, Inflarx, Janssen-Cilag, Leo Pharma, Novartis, Regeneron and Sanofi, for participation as an investigator, and received speaker honoraria from AbbVie, Boehringer-Ingelheim, Galderma and MSD. He has also received unrestricted departmental grants from Abbvie, Leo Pharma and Novartis. G. Damiani has received honoraria from Novartis and Galderma for participation on advisory boards, and grants from Almirall and Rocchetta for participation as an investigator, and speaker honoraria from Novartis and Sanofi.

Ethics Approval

IRB status: IRB00224032 approved by John Hopkins University Ethical Committee.

Informed Consent

All patients signed an informed consent form before being enrolled in this study.

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