

Gender Differences in Hidradenitis Suppurativa

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OBJECTIVE.

Examine gender differences in risk factors, disease manifestations, and comorbidities in hidradenitis suppurativa (HS).

TAKEAWAY.

Our study found significant gender differences in HS epidemiology, disease presentation, and cutaneous comorbidities. Pathophysiology potentially underlying gender differences in HS, including hormonal influences, warrants further investigation.

JLH is on the Board of Directors for the Hidradenitis Suppurativa Foundation and has served as an advisor for Novartis and a speaker for AbbVie. VYS is on the Board of the Directors for the Hidradenitis Suppurativa Foundation, and has served as an advisor, investigator and/or speaker for Sanofi Genzyme, Regeneron, AbbVie, Burt's Bees, Dermira, Eli Lilly, Novartis, Pfizer, Galderma, Leo Pharma, SUN Pharma, Menlo Therapeutics, GpSkin, and Skin Actives Scientific. TS, JS, SM, CH, AL, RB report no conflicts of interest. There was no financial transaction for the preparation of this manuscript.

Background and Objective

- Current literature on HS in the United States and in Europe have identified a predominance of women and increased disease severity in men, suggesting possible gender differences underlying the pathophysiology of HS.^{1,2}
- However, few studies have characterized gender differences at length.
- The goal of this project is to examine gender differences in risk factors, disease manifestations, and comorbidities in HS through a retrospective cohort study at a single academic center.

Methods

- A retrospective chart review gathered demographic, disease, and comorbidity data from all HS patients who presented to the UCLA HS clinic between August 2009 and March 2018.
- Comparative analyses between genders were performed using Wilcoxon rank sum and Fisher's exact test.
- Logistic regression calculated gender differences in risk of presenting with severe disease (Hurley-3 v Hurley-1 and Hurley-2) and mean Dermatology Life Quality Index (DLQI) after controlling for Hurley stage.
- $P < 0.05$ was considered statistically significant.

Results

- Our cohort of 209 patients (140 women, 69 men) was racially diverse (28% White, 72% non-White).

Demographics and disease characteristics	Total (n=209)	Women (n=140)	Men (n=69)	P-value
Race/ethnicity (n=209)				0.3304 [^]
White	59 (28%)	43 (31%)	16 (23%)	
Non-White	150 (72%)	97 (69%)	53 (77%)	
Hispanic	35 (17%)	22 (16%)	13 (19%)	
Black	34 (16%)	29 (21%)	5 (7%)	
Asian	23 (11%)	10 (7%)	13 (19%)	
Middle Eastern	13 (6%)	5 (4%)	8 (12%)	
Native American	3 (1%)	2 (1%)	1 (1%)	
Bi- or multi-racial	29 (14%)	22 (16%)	7 (10%)	

[^]Fischer's exact test comparing proportions of White vs non-White patients between genders

Results

- Women were more likely to have earlier disease onset (23 vs 25 years, $p=0.046$).
- Men had 2.24x risk of presenting with Hurley-3 ($p=0.004$).

Demographics and disease characteristics	Total (n=209)	Women (n=140)	Men (n=69)	P-value
Age at presentation to HS clinic, mean \pm SD (range) (n=209)	34 \pm 12 (15-75)	34 \pm 13 (15-75)	34 \pm 11 (17-62)	0.94
Duration of disease, mean \pm SD (range) (n=197)	11 \pm 9 (0-50)	12 \pm 10 (0-50)	9 \pm 8 (0-30)	0.066
Age of HS onset, mean \pm SD (range) (n=197)	23 \pm 12 (6-63)	23 \pm 12 (6-63)	25 \pm 11 (9-60)	0.046*
BMI, mean \pm SD (range) (n=192)	31 \pm 8 (19-60)	31 \pm 8 (19-50)	31 \pm 9 (19-60)	0.93
Family history of HS (n=195)	55 (28%)	41 (31%)	14 (23%)	0.31
Active smoker (n=208)	35 (17%)	16 (12%)	19 (28%)	0.005*
Former smoker (n=195)	44 (25%)	31 (25%)	13 (26%)	1.00
Disability status (n=146)	20 (10%)	13 (10%)	7 (11%)	0.80
Severity of HS (n=209)				0.011*&
Hurley stage 1	70 (33%)	53 (38%)	17 (25%)	
Hurley stage 2	99 (47%)	68 (49%)	31 (45%)	
Hurley stage 3	40 (19%)	19 (14%)	21 (30%)	
Initial DLQI score, mean \pm SD (range) (n=132)	14 \pm 9 (0-30)	14 \pm 9 (0-30)	14 \pm 9 (0-30)	0.81

Abbreviations: BMI, body mass index; DLQI, Dermatologic Life Quality Index; HS, hidradenitis suppurativa; SD, standard deviation

*Statistically significant ($p \leq 0.05$)

&Fischer's exact test comparing proportions of patients with Hurley 1, 2, or 3 disease between genders

Results

- Men had greater gluteal (52% vs 25%, $p=0.002$) involvement versus greater breasts/chest involvement in women (18% vs 4%, $p=0.008$).
- Men were significantly more likely to have posterior neck (6% vs 1%, $p=0.042$) or scalp (6% vs 1%, $p=0.042$) involvement.

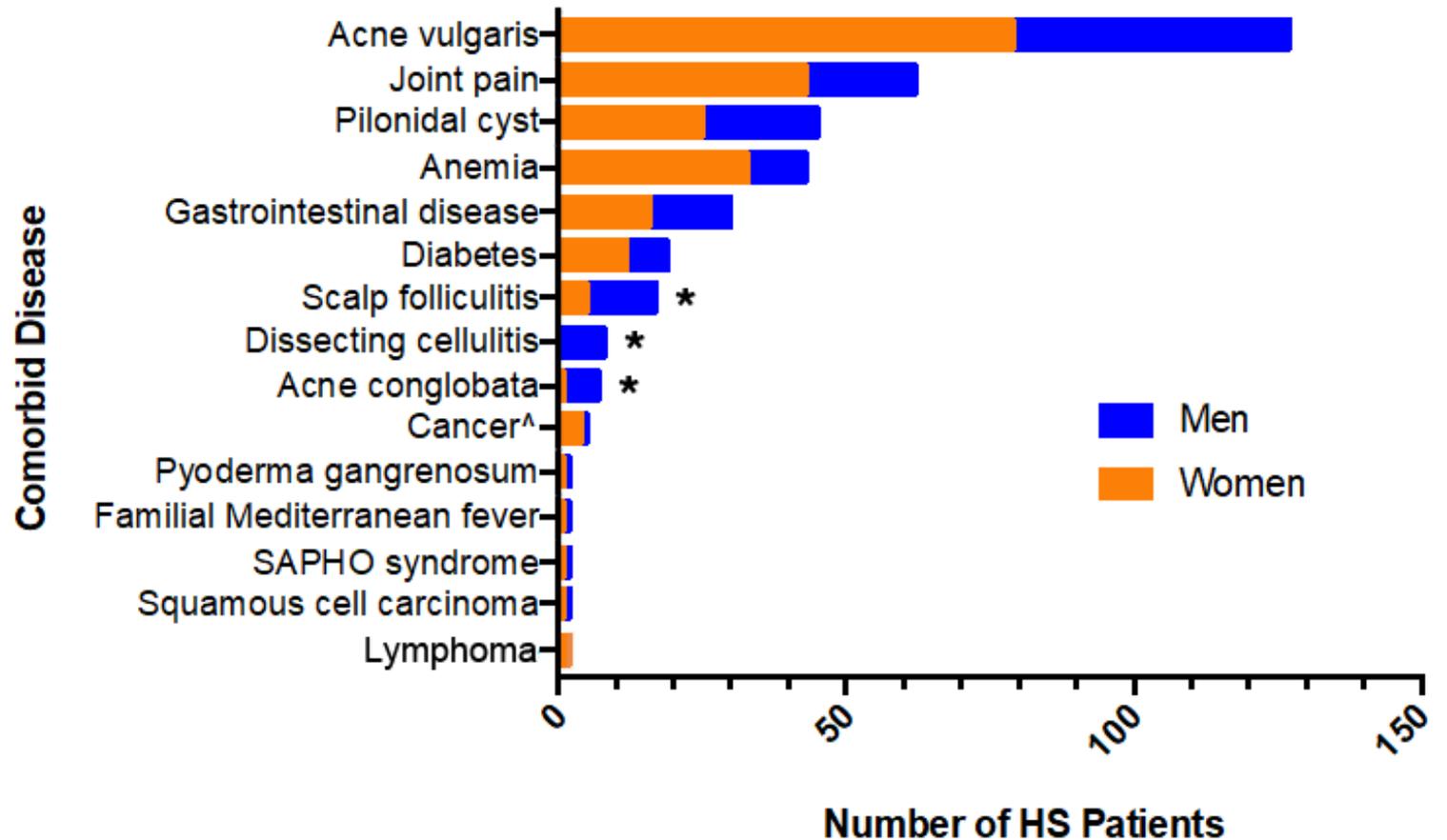
Location of HS	Total (n=209)	Women (n=140)	Men (n=69)	P-value
Axilla	131 (63%)	89 (64%)	42 (61%)	0.76
Groin (including genitals)	103 (49%)	68 (49%)	35 (51%)	0.88
Gluteal (including perianal)	71 (34%)	35 (25%)	36 (52%)	0.0002*
Inner thigh	57 (27%)	40 (29%)	17 (25%)	0.62
Breast/chest	28 (13%)	25 (18%)	3 (4%)	0.008*
Perineal	4 (2%)	1 (1%)	3 (4%)	0.11
Back of neck	5 (2%)	1 (1%)	4 (6%)	0.042*
Scalp	5 (2%)	1 (1%)	4 (6%)	0.042*
Face	2 (1%)	0 (0%)	2 (3%)	0.11

*Statistically significant ($p \leq 0.05$)

Results

Men were disproportionately affected by:

- Acne conglobata (11% vs 1%, $p=0.004$)
- Scalp folliculitis (21% vs 4%, $p=0.0005$)
- Dissecting cellulitis (12% vs 0%, $p=0.0001$).



Abbreviations: HS, hidradenitis suppurativa; SAPHO, Synovitis, Acne, Pustulosis, Hyperostosis, Osteitis

* $p \leq 0.05$, indicating statistically significant difference between genders

^Excluding skin cancers

Conclusions

- Women had earlier onset of disease compared to men
- Men had increased risk of presenting with severe HS
- HS generally affected frontal anatomic regions in women versus posterior regions in men, supporting prior studies.^{1,3}
- Further studies may explore if patients with atypical HS have increased risk of misdiagnosis or differential treatment responses compared to those with classic intertriginous presentations.
- More men have comorbid acne conglobata, dissecting cellulitis, and pilonidal cyst, consistent with male predominance of these conditions overall.⁴

Conclusions

- Our racially diverse cohort demonstrates significant gender differences in epidemiology, cutaneous comorbidities, and disease manifestations of HS.
- Future investigations may consider gender-tailored treatments by exploring mechanisms underlying gender differences in HS, from hormonal influences to immune profiles.

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