

Article



Hematologic Malignancy Prevalence in Hidradenitis Suppurativa: A Cross-Sectional Study

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Abstract: Background: Hidradenitis suppurativa (HS) is a chronic inflammatory skin disease associated with substantial comorbidity. Evidence regarding the co-occurrence of HS and recorded hematologic malignancy diagnoses in large, diverse electronic health record populations remains limited. Methods: We conducted a cross-sectional analysis using standardized electronic health record data from the All of Us Research Program. Adults aged 18–90 years with available electronic health records were included. Participants with HS were identified using ICD-10-CM and SNOMED diagnostic codes matched to controls without recorded HS in a 1:4 ratio using nearest-neighbor propensity-score. Matching was based on age, gender, and race/ethnicity. Matching was performed without replacement using a caliper of 0.2 standard deviations of the logit of the propensity score. Multivariable logistic regression models with cluster-robust standard errors for matched sets were used to evaluate associations between HS and recorded hematologic malignancy diagnoses. Results: The matched cohort included 2636 participants with HS and 10,544 controls. The primary complete-case analysis included 1544 participants with HS and 6176 controls. Any recorded hematologic malignancy was identified in 72 participants with HS (4.66%) and 151 controls (2.44%) and was associated with HS after covariate adjustment (adjusted odds ratio [aOR], 2.04; 95% CI, 1.62–2.47). Recorded diagnoses of multiple myeloma (22 [1.42%] vs. 25 [0.40%]; aOR, 3.64; 95% CI, 2.29–6.24), leukemia (24 [1.55%] vs. 41 [0.66%]; aOR, 2.30; 95% CI, 1.46–3.82), and malignant lymphoma (39 [2.53%] vs. 95 [1.54%]; aOR, 1.83; 95% CI, 1.30–2.57) were also more frequently recorded among participants with HS. Conclusions: HS was associated with increased adjusted odds of recorded hematologic malignancies in this large, diverse cohort. Longitudinal studies are needed to establish temporality and causality.

Keywords: hidradenitis suppurativa; hematologic malignancies; autoimmune disease; chronic inflammation; epidemiology



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1. Introduction

Hidradenitis suppurativa (HS) is a chronic relapsing inflammatory skin disease characterized by painful nodules, abscesses, draining tunnels, and scarring in intertriginous regions [1,2]. Although once viewed primarily as a localized cutaneous disorder, HS is now increasingly recognized as a systemic inflammatory disease with substantial comorbidity burden extending beyond the skin [3,4]. Epidemiologic studies and reviews have linked HS with metabolic, cardiovascular, endocrine, gastrointestinal, rheumatologic, and psychiatric comorbidities, supporting the concept that HS reflects broader immune dysregulation rather than an isolated follicular disorder [5,6].

The relationship between chronic inflammatory disease and malignancy has attracted increasing attention because persistent antigenic stimulation, cytokine-driven immune activation, impaired immune surveillance, and exposure to immunomodulatory therapies may all contribute to oncogenesis [7]. In systemic autoimmune and inflammatory conditions such as rheumatoid arthritis, systemic lupus erythematosus, and Sjögren syndrome, increased risks of lymphoid and other hematologic malignancies have been repeatedly described [8,9]. Large population-based analyses have shown that several autoimmune conditions are associated with increased risk of non-Hodgkin lymphoma, and reviews of connective tissue diseases likewise describe clinically important links between chronic immune dysregulation and hematologic cancer [9,10]. These observations provide a biologically plausible framework for investigating whether similar patterns may be present in HS.

Emerging data suggest that HS may also be associated with malignancy. Prior studies have reported increased overall cancer risk in HS, with elevated risks for several specific cancers including nonmelanoma skin cancer, prostate cancer, and Hodgkin lymphoma [11], while systematic reviews have also highlighted the risk of malignant degeneration of chronic HS lesions into cutaneous squamous cell carcinoma in severe longstanding disease [12]. However, despite this growing literature, large-scale epidemiologic studies specifically examining hematologic malignancies in HS remain limited [13,14]. The strength and consistency of these associations have not been fully characterized across diverse populations [13,14]. Given the chronic inflammatory nature of HS and the established relationship between immune-mediated disease and hematologic neoplasia, further study of hematologic malignancy prevalence is warranted. Therefore, this study aimed to evaluate the prevalence of hematologic malignancies among patients with HS.

2. Methods

2.1. Study Design and Data Source

This study was a cross-sectional analysis conducted in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guidelines [15]. The study utilized standardized data from the All of Us (AoU) Research Program. AoU is an ongoing National Institutes of Health initiative designed to create a large and diverse biomedical research database by enrolling populations historically underrepresented in research [16]. As of November 2024, the AoU dataset includes data from more than 800,000 participants, representing a socioeconomically and demographically diverse cohort with a balanced gender distribution, a wide age range, and substantial representation from multiple racial and ethnic groups (18.65% Black, 15.65% Hispanic, and 3.35% Asian). The program integrates multiple sources of health information including electronic health records (EHRs), self-reported surveys, physical measurements, biospecimens, and digital health device data.

Data within the AoU Research Program are mapped using the Observational Health and Medicines Outcomes Partnership (OMOP) common data model v5.2, which standardizes clinical data across participating health systems and enables consistent data extraction and analysis across multiple sites. The Institutional Review Board of the AoU Research Program approved the study protocol and waived the requirement for informed consent for this analysis. Access to medical record data was granted and all data used in this study was de-identified to protect patient privacy. All research procedures were conducted in accordance with the principles outlined in the Declaration of Helsinki [17].

2.2. Participant Population

Participants were selected from the AoU Research Program database. Inclusion criteria consisted of individuals aged 18–90 years at the time of enrollment who had available EHR data within the AoU Research Program dataset. Cases consisted of individuals diagnosed with HS identified using the International Classification of Diseases, 10th Revision, Clinical Modification (ICD-10-CM) code L73.2 and the Systematized Nomenclature of Medicine (SNOMED) code 59393003 within the available EHR data. Control participants without a diagnosis of HS were selected from the same database. HS patients were matched with controls in a 1:4 ratio using nearest

neighbour matching without replacement and a caliper of 0.2 standard deviations of the logit of the propensity score, based on gender, age, and race/ethnicity [18].

This ratio matching strategy was selected to minimize baseline demographic differences between groups, enhance the precision of effect sizes without overfitting, and to improve comparability between HS patients and controls [19]. The matched-set identifier was retained for use in the regression analyses. The participant selection process and cohort construction are illustrated in Figure 1.

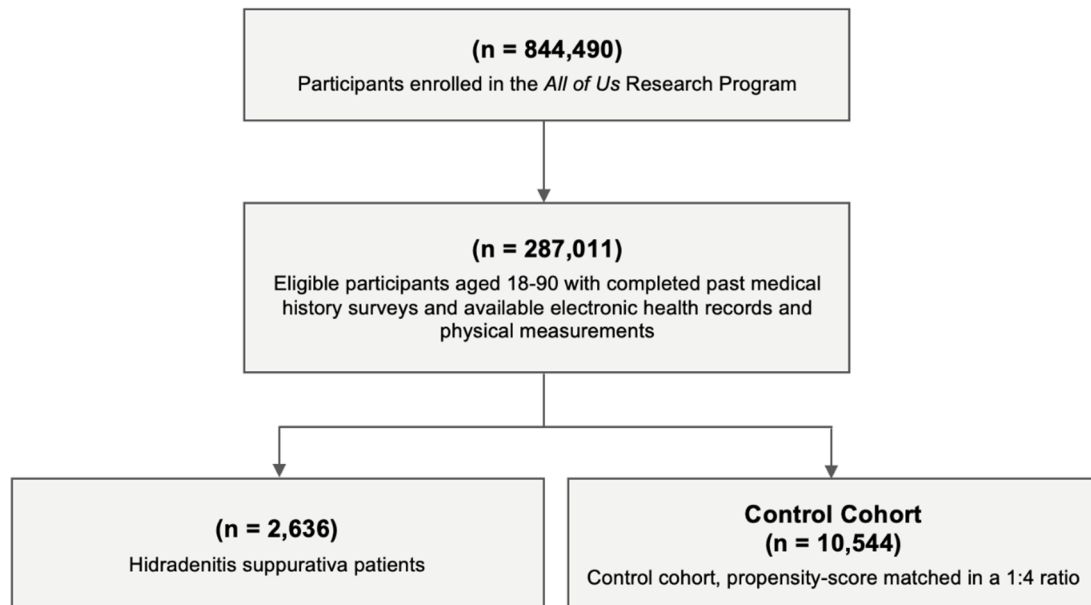


Figure 1. Years with completed medical history surveys, available electronic health records, and physical measurements were identified. Individuals with hidradenitis suppurativa (HS) were matched to control participants in a 1:4 ratio using propensity score matching based on age, gender, and race/ethnicity.

2.3. Variables and Data Extraction

The primary outcome was the presence of any recorded hematologic malignancy diagnosis among participants with HS compared with matched controls. Secondary outcomes were recorded diagnoses of multiple myeloma, leukemia, and malignant lymphoma. HS and hematologic malignancies were identified using diagnostic codes mapped to standardized concepts in the Observational Medical Outcomes Partnership common data model. The available SNOMED codes and OMOP concept definitions are reported in Supplementary Table S1. Absence of an included diagnostic code was classified as absence of the corresponding recorded diagnosis. Diagnoses were not independently validated through chart review, pathology records, or linkage to a cancer registry.

Additional variables included age, gender, race/ethnicity, annual income, education level, smoking status, body mass index, type 2 diabetes, hypertension, dyslipidemia, selected autoimmune diseases, and coded immunosuppression. The selected autoimmune diseases included psoriasis, rheumatoid arthritis, systemic lupus erythematosus, inflammatory bowel disease, and type 1 diabetes. These variables were obtained from standardized AoU data, including electronic health records, participant surveys, and physical measurements, as applicable. Coded immunosuppression was determined from EHR diagnostic codes and should not be interpreted as a measure of biologic or systemic treatment exposure.

Given that this study used cross-sectional EHR-derived diagnosis data, temporal ordering between HS and hematologic malignancy could not be reliably established. Recorded diagnosis dates may reflect clinical documentation, billing activity, or imported medical history and do not necessarily represent true disease onset, first clinical diagnosis, or complete longitudinal history. Accordingly, hematologic malignancies were analyzed as recorded prevalent diagnoses rather than as incident outcomes following HS diagnosis.

2.4. Statistical Analysis

Comparative statistical analyses were performed utilizing the Pearson χ^2 test for categorical variables and Fisher's exact test when expected cell counts were low. Continuous variables were compared using the unpaired t-test. Univariable logistic regression was initially performed to evaluate crude associations between HS and

recorded hematologic malignancy diagnoses. Subsequently, multivariable logistic regression was used to calculate the adjusted odds ratios (aOR) and corresponding 95% confidence intervals (CIs) for the prevalence of hematologic malignancy diagnoses among patients with HS compared with matched controls. Multivariable models were adjusted for age, gender, race/ethnicity, annual income, education, smoking status, body mass index, type 2 diabetes, hypertension, dyslipidemia, selected autoimmune diseases, and coded immunosuppression. The selected autoimmune diseases included psoriasis, rheumatoid arthritis, systemic lupus erythematosus, inflammatory bowel disease, and type 1 diabetes. As participants were selected using 1:4 propensity score matching, regression models accounted for matched set membership using cluster-robust standard errors by matched set.

Missingness was assessed for all covariates, and no multiple imputation was performed. The primary adjusted analyses used complete-case analysis at the matched-set level. An entire matched set was excluded when a required smoking or body mass index value was unavailable for any member. The resulting primary adjusted sample included 1544 matched sets and 7720 participants. Nonresponse categories for annual income and education were retained as separate analytical categories. Model-specific sample sizes and event counts are reported in Supplementary Table S2. As a sensitivity analysis, the models were repeated in the full matched cohort using separate missing-data categories for smoking status and body mass index; these results are reported in Supplementary Table S3. Statistical significance was defined as a two-sided *p* value of less than 0.05, with 95% CIs calculated using the Wald method.

All statistical analyses were performed using the R software (version 4.3.1; R foundation for Statistical Computing, Vienna, Austria) within the AoU Researcher Workbench, a secure cloud-based platform designed to facilitate controlled access to the AoU Research Program data [20]. The Researcher Workbench provides an integrated Jupyter Notebook environment that supports reproducible statistical analysis using standardized datasets mapped to the OMOP common data model. Cohort construction, dataset generation, and analytic code used in this study are documented within the AoU Researcher Workbench and are available to authorized researcher through the AoU platform or from corresponding author upon reasonable request.

3. Results

3.1. Demographic and Clinical Characteristics

From the 844,490 participants enrolled in the AoU Research Program, 2636 patients with HS and 10,544 controls were identified. Following propensity score matching at a 1:4 ratio based on age, gender, and race/ethnicity, the final analytic cohort consisted predominantly of female participants (80.54%). The racial and ethnic composition of the cohort was diverse with 49.28% of participants identifying as Black or Hispanic. Baseline sociodemographic and clinical characteristics of the HS and control cohorts are summarized in Table 1. Compared with matched controls, patients with HS demonstrated higher prevalence of several clinical and lifestyle risk factors. Current or prior smoking was more frequently observed among individuals with HS, consistent with prior epidemiologic studies of the disease. Patients with HS also exhibited higher mean body mass index relative to control participants. Differences were also observed across socioeconomic indicators, including annual household income and education level, reflecting the broad demographic representation within the AoU dataset. Smoking and body mass index data were unavailable for a substantial proportion of the cohort. The primary adjusted analyses therefore used complete matched sets. The resulting model-specific sample sizes and event counts are reported in Supplementary Table S2. Sensitivity analyses using separate missing-data categories for smoking status and body mass index are reported in Supplementary Table S3.

Table 1. Sociodemographic and clinical traits of hidradenitis suppurativa cases/controls in the All of Us Research Program.

Characteristic	Cases No. (%) (n = 2636)	Controls No. (%) (n = 10,544)	<i>p</i> Value
Age (years old) [†]			>0.99
18–39	852 (32.32)	3408 (32.32)	
40–59	1129 (42.83)	4516 (42.83)	
60–74	559 (21.21)	2236 (21.21)	
≥75	96 (3.64)	384 (3.64)	
Gender [†]			>0.99
Male	489 (18.55)	1956 (18.55)	
Female	2123 (80.54)	8492 (80.54)	
Other ¹	24 (0.91)	96 (0.91)	

Table 1. Cont.

Characteristic	Cases No. (%) (n = 2636)	Controls No. (%) (n = 10,544)	p Value
Race/Ethnicity [†]			>0.99
White	1114 (42.26)	4456 (42.26)	
Black	914 (34.67)	3656 (34.67)	
Hispanic	385 (14.61)	1540 (14.61)	
Other ²	223 (8.46)	892 (8.46)	
Annual Income			<0.001
≥\$50k	685 (25.99)	3665 (34.76)	
\$35k–\$50k	255 (9.67)	882 (8.36)	
\$25k–\$35k	238 (9.03)	835 (7.92)	
\$10k–\$25k	447 (16.96)	1305 (12.38)	
≤\$10k	522 (19.80)	1808 (17.15)	
Other ³	489 (18.55)	2049 (19.43)	
Education			<0.001
College graduate	776 (29.44)	4061 (38.51)	
Attended college, no degree	967 (36.68)	2956 (28.03)	
12th Grade	601 (22.80)	2257 (21.41)	
No HS degree	224 (8.50)	1004 (9.52)	
Other ³	68 (2.58)	266 (2.52)	
Malignancy Outcomes			<0.001
Multiple myeloma	36 (1.37)	41 (0.39)	
Leukemia	39 (1.48)	69 (0.65)	
Malignant lymphoma	64 (2.43)	163 (1.55)	
Ever smoker ⁴	996/1652 (60.29)	3160/6608 (47.82)	<0.001
Average BMI ⁵ (SD)	37.83 (8.60)	30.87 (9.48)	<0.001

[†] Indicates variable propensity matched with control cohort. ¹ Includes nonbinary, transgender, and other gender identities. ² Includes Middle Eastern or North African, American Indian or Alaska Native, Hawaiian or another Pacific Islander, and other race and ethnicity. ³ Includes participants who preferred not to answer the question. ⁴ Smoking percentages were calculated among participants in complete matched sets with available smoking information. ⁵ BMI values were calculated among participants in complete matched sets with available BMI information. Ref: reference, No: Number, SD: standard deviation, HS: high school, BMI: body mass index.

3.2. Prevalence of Hematologic Malignancies

The primary multivariable analysis was performed in the complete-case matched sample, comprising 1544 matched sets comprising 1544 participants with HS and 6176 controls. Within this analytic sample, 72 participants with HS (4.66%) and 151 controls (2.44%) had at least one recorded hematologic malignancy (Supplementary Table S2). These counts differ from the descriptive full-cohort counts presented in Table 1 because the adjusted analysis excluded matched sets with missing smoking or BMI data. After adjustment for measured demographic, socioeconomic, lifestyle, and clinical covariates, HS was associated with higher odds of a recorded hematologic malignancy diagnosis (aOR, 2.04; 95% CI, 1.62–2.47; Table 2).

Table 2. Univariable and multivariable-adjusted odds of hematologic malignancies in patients with hidradenitis suppurativa.

Outcome	Univariable OR (95% CI)	p Value	Multivariable aOR (95% CI) [†]	p Value
Hematologic malignancy ¹	1.86 (1.47, 2.34)	<0.001	2.04 (1.62, 2.47)	<0.001
Multiple myeloma	3.55 (2.28, 5.95)	<0.001	3.64 (2.29, 6.24)	<0.001
Leukemia	2.28 (1.38, 3.71)	<0.001	2.30 (1.46, 3.82)	<0.001
Malignant lymphoma	1.58 (1.08, 2.26)	0.0156	1.83 (1.30, 2.57)	0.0025
Race/Ethnicity ¹				
White	1.89 (1.28, 3.01)	0.011	1.92 (1.34, 3.09)	0.014
Black	2.12 (1.48, 3.41)	0.008	2.25 (1.57, 3.68)	0.004
Hispanic	3.65 (2.10, 6.78)	<0.001	3.82 (1.98, 7.03)	<0.001
Gender ¹				
Male	2.12 (1.51, 3.47)	<0.001	2.45 (1.73, 3.35)	<0.001
Female	1.77 (1.30, 3.01)	<0.001	1.99 (1.52, 3.30)	<0.001

aOR: multivariable adjusted odds ratio, CI: confidence interval. [†] aOR: Multivariable regression analysis controlled for age, race, ethnicity, gender, income, education, body mass index, type 2 diabetes, hypertension, dyslipidemia, autoimmune diseases

(including psoriasis, rheumatoid arthritis, systemic lupus erythematosus, inflammatory bowel disease, and type 1 diabetes), immunosuppression, and smoking status. ¹ Includes patients with non-Hodgkin's lymphoma, malignant lymphoma of lymph nodes, mantle cell lymphoma, Hodgkin's disease, lymphosarcoma, acute lymphoid leukemia, chronic lymphoid leukemia, acute myeloid leukemia, chronic myeloid leukemia, myeloproliferative neoplasms, monocytic leukemia, plasma cell leukemia, subacute leukemia, and multiple myeloma.

3.3. Association Between HS and Specific Hematologic Malignancies

In the same complete-case analytic sample, multiple myeloma was recorded in 22 participants with HS (1.42%) and 25 controls (0.40%; aOR, 3.64; 95% CI, 2.29–6.24). Leukemia was recorded in 24 participants with HS (1.55%) and 41 controls (0.66%; aOR, 2.30; 95% CI, 1.46–3.82). Malignant lymphoma was recorded in 39 participants with HS (2.53%) and 95 controls (1.54%; aOR, 1.83; 95% CI, 1.30–2.57) (Table 2; Supplementary Table S2). These estimates describe cross-sectional associations with recorded diagnoses and do not establish temporal sequence. Adjusted odds ratios for hematologic malignancies among patients with HS compared with matched controls are illustrated in Figure 2.

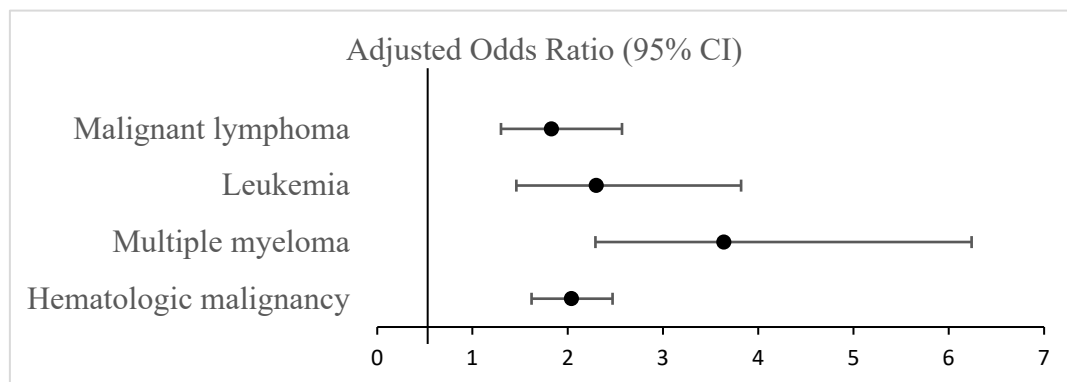


Figure 2. Forest plot showing adjusted odds ratio for hematologic malignancies among patients with hidradenitis suppurativa with matched controls. Points represent adjusted odds ratios derived from multivariable logistic regression model, and horizontal lines represent 95% confidence intervals. Models were adjusted for age, gender, race/ethnicity, socioeconomic status, smoking status, body mass index, immunosuppression, and autoimmune comorbidities. The vertical reference line indicates an odds ratio of 1.

3.4. Stratified Analyses by Race and Gender

Exploratory stratified analyses by race/ethnicity and gender are presented in Table 2. Among Hispanic participants, HS was associated with higher odds of a recorded hematologic malignancy diagnosis compared with matched controls (aOR, 3.82; 95% CI, 1.98–7.03; $p < 0.001$). However, formal interaction testing was not performed; therefore, differences among subgroup-specific estimates should not be interpreted as evidence that the association varied by race/ethnicity or gender. Stratified analyses by race/ethnicity and gender are presented in Table 2, with corresponding forest plots shown in Figures 3 and 4, respectively.

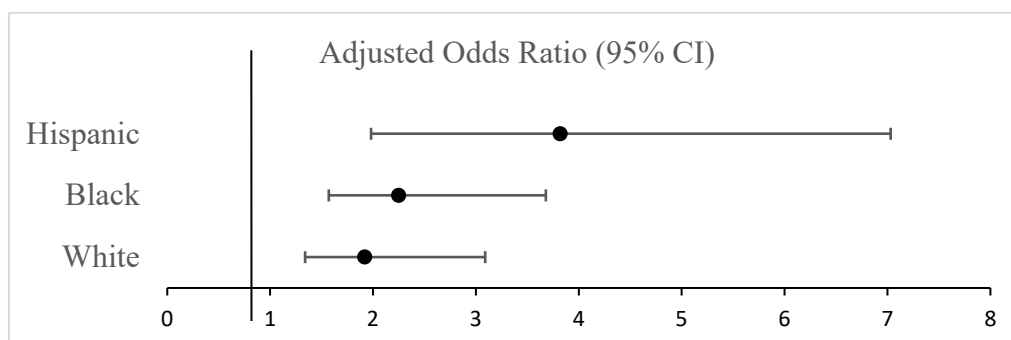


Figure 3. Forest plot showing adjusted odds ratios for hematologic malignancies among patients with hidradenitis suppurativa compared with matched controls, stratified by race and ethnicity. Points represent adjusted odds ratios derived from multivariable logistic regression models, and horizontal lines represent 95% confidence intervals. Models were adjusted for age, gender, socioeconomic status, smoking status, body mass index, immunosuppression, and autoimmune comorbidities. The vertical reference line indicates an odds ratio of 1.

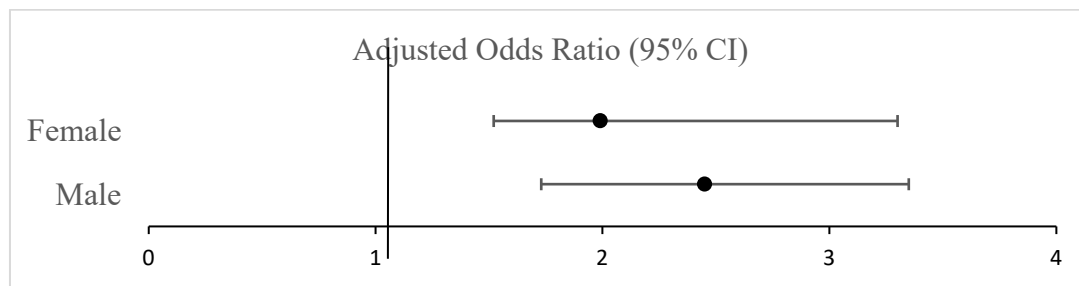


Figure 4. Forest plot showing adjusted odds ratios for hematologic malignancies among patients with hidradenitis suppurativa compared with matched controls, stratified by gender. Points represent adjusted odds ratios derived from multivariable logistic regression models, and horizontal lines represent 95% confidence intervals. Models were adjusted for age, race/ethnicity, socioeconomic status, smoking status, body mass index, immunosuppression, and autoimmune comorbidities. The vertical reference line indicates an odds ratio of 1.

4. Discussion

In this large cross-sectional analysis of AoU EHR data, recorded hematologic malignancy diagnoses were more prevalent among participants with HS than among matched controls. Strong associations were observed for multiple myeloma, leukemia, and malignant lymphoma after adjustment for selected measured covariates. Our findings are consistent with prior epidemiologic studies linking HS with lymphoma and increased malignancy risk [13,14], while further extending this literature by demonstrating significant associations across multiple hematologic cancer subtypes within a diverse EHR cohort. However, the present analysis cannot determine whether HS preceded hematologic malignancy or whether the observed associations reflect causation, shared risk factors, treatment effects, differential healthcare contact, incomplete observation, or diagnostic misclassification. In a population-based cohort analysis conducted in the United States, Tannenbaum et al. reported that patients with HS had approximately 2-to-4-fold higher odds of lymphoma overall, and that the association varied across gender and age subgroups [13]. A separate nationwide study by Jung et al. found that HS was associated with increased overall cancer risk and significantly increased risk of Hodgkin lymphoma, with stronger associations in some sex-stratified analyses and in patients with more severe disease [14]. More recently, systematic reviews have found that patients with HS appear to have higher cancer risk than the general population, with recurrent signals for squamous cell carcinoma, lymphoma, and several internal malignancies [12,21]. In that context, our findings are concordant with the existing lymphoma signal in HS and add granularity by identifying associations with leukemia and multiple myeloma.

The growing body of literature suggests that lymphoproliferative risk in HS is not incidental. Several biologic mechanisms may be hypothesized to provide context for the observed associations. Chronic inflammation has been linked to sustained cytokine signaling, oxidative stress, impaired immune surveillance, and cumulative cellular damage. In HS, studies of lesional tissue have identified substantial B-cell and plasmablast involvement, suggesting that B-cell dysregulation may warrant further investigation in relation to plasma cell and lymphoid disorders [22,23]. Alongside the well-characterized Th17/IL-17 axis, HS lesions have demonstrated elevated B-cell-activating factor, ectopic germinal centers, and plasmablast expansion [24,25]. Other proposed inflammatory pathways in HS include IL-36 γ expression, NLRP3 inflammasome activation, neutrophil-derived reactive oxygen species, and dysregulated STAT3/JAK signaling [26]. However, the present study did not evaluate inflammatory biomarkers, immune-cell populations, somatic mutations, clonal hematopoiesis, or treatment effects. These pathways therefore represent hypotheses for future investigation and should not be interpreted as mechanisms established by our findings.

Our study also contributes to the growing understanding of HS as a systemic inflammatory disease rather than an exclusively cutaneous condition. Prior literature has described the substantial inflammatory and comorbidity burden associated with HS, including cardiometabolic, psychiatric, and immune-mediated conditions [27,28]. The present findings extend this literature by identifying an association between HS and recorded prevalent hematologic malignancy diagnoses. As temporal ordering could not be established, these findings should not be interpreted as demonstrating incident malignancy risk or causation. The subgroup estimate among Hispanic participants was notable but exploratory. As formal interaction testing was not performed, it cannot be concluded that the association differed by race or ethnicity, and this observation requires replication in other datasets.

This study has several strengths. First, the study utilized data from the AoU Research Program, a large multi-site database specifically designed to recruit populations historically underrepresented in biomedical research, thereby increasing the demographic diversity and real-world representativeness of the cohort. Second, the matched

design improved comparability on the measured matching variables, while multivariable adjustment accounted for selected measured. Third, the inclusion of specific hematologic malignancy subtypes allowed more detailed characterization of the association than prior HS cancer studies that focused primarily on overall cancer risk or lymphoma. Finally, the racial and ethnic diversity of this cohort is an important strength, given that many earlier HS epidemiologic studies have been conducted in more homogenous national datasets [11].

This study has several limitations. First, HS, hematologic malignancies, and comorbidities were defined using recorded EHR diagnostic codes and were not independently validated through chart review, pathology records, or linkage to a cancer registry. Absence of a diagnostic code was classified as absence of the recorded condition, which may have produced under-ascertainment. Second, the cross-sectional design cannot establish whether HS preceded hematologic malignancy, and reverse causality cannot be excluded. Recorded diagnosis dates may reflect billing activity, imported medical history, or incomplete care across contributing health systems rather than true disease onset. Third, the available analysis did not include validated measures of HS severity, disease duration, cumulative inflammatory burden, or detailed biologic and systemic treatment exposure. Fourth, differences in healthcare utilization and duration of observable EHR data may have produced detection bias. Fifth, smoking and body mass index were unavailable for a substantial proportion of the cohort, and complete-case analysis may have introduced selection bias. Binary smoking adjustment may also leave residual confounding related to smoking duration and intensity. We could not perform additional phenotype, observation-time, healthcare-utilization, interaction, or alternative propensity-score sensitivity analyses using the available analytic materials. Finally, subgroup analyses had smaller event counts and should be regarded as exploratory. These limitations preclude causal interpretation, estimation of incident malignancy risk, and recommendations regarding hematologic cancer screening.

Future studies should use longitudinal cohorts with validated HS and malignancy phenotypes, comparable periods of healthcare observation, detailed treatment exposure, and sufficient follow-up to evaluate incident outcomes. Linkage to cancer registries and incorporation of smoking intensity, HS severity, and treatment duration would help distinguish shared risk factors, treatment effects, detection bias, and possible biological associations.

5. Conclusions

In this cross-sectional analysis of participants in the All of Us Research Program, HS was associated with higher odds of recorded prevalent hematologic malignancy diagnoses, including multiple myeloma, leukemia, and malignant lymphoma, compared with matched controls. The association remained after adjustment for measured demographic, socioeconomic, lifestyle, and clinical covariates. These findings expand the literature concerning the systemic comorbidity burden associated with HS. However, temporal sequence and causality cannot be established from the available EHR-derived data. Longitudinal studies incorporating validated cancer outcomes, treatment exposure, healthcare utilization, and more detailed smoking information are needed to determine whether HS is independently associated with the subsequent development of hematologic malignancy.

Supplementary Materials

The additional data and information can be downloaded at: <https://media.sciltp.com/articles/others/2609291756144741/DSS-26030129-SI.pdf>.

Author Contributions

P.A.: conceptualization, methodology, software, formal analysis, investigation, data curation, project administration, and writing—original draft preparation. Z.H., M.J.D. and S.E.: methodology, formal analysis, and validation of the statistical analyses. M.R. and E.M.: writing—review and editing. J.A., R.G. and D.C.: conceptualization, supervision, and writing—review and editing. All authors have read and agreed to the submitted version of the manuscript and agree to be accountable for their contributions.

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Institutional Review Board Statement

Not applicable.

Informed Consent Statement

Not applicable.

Data Availability Statement

The data analyzed in this study are available to approved researchers through the All of Us Researcher Workbench, subject to the program's registration, data-use, and dissemination policies. Participant-level data cannot be publicly shared or provided with the manuscript. The analytic code is documented in the All of Us Researcher Workbench and is available to authorized researchers through the platform or from the corresponding author upon reasonable request, subject to All of Us policies.

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Conflicts of Interest

David Croitoru has received consultation fees and educational grants from AbbVie, Amgen, Arcutis, Bausche Health, BioJAMP, Boehringer-Ingelheim, Bristol-Myers-Squibb, Eli-Lilly, Janssen, Novartis, Pfizer, Sanofi-Regeneron, Sun Pharma and UCB. Parsa Abdi is an Editorial Board Member, and Robert Gniadecki is Editor-in-Chief of Dermatology and Skin Science. Given their respective roles, Parsa Abdi and Robert Gniadecki had no involvement in the peer review of this paper and had no access to information regarding its peer-review process. Full responsibility for the editorial process was delegated to another editor of the journal. The other authors declare no conflict of interest.

Use of AI and AI-Assisted Technologies

No AI tools were utilized for this paper.

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