REVIEW

The Correlation between Hidradenitis Suppurativa and Irritable Bowel Diseases: A Systematic Review

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J Clin Aesthet Dermatol. 2025;18(5):20–25.

BACKGROUND: Hidradenitis suppurativa (HS) is an inflammatory skin condition that presents with nodules or abscesses associated with sinus tracts. Inflammatory bowel diseases (IBD) are inflammatory gastrointestinal (GI) conditions presenting with irregular bowel movements. Although HS and IBDs may have similarities in their clinical presentation and pathophysiology, they can differ in their cutaneous manifestations. Both conditions are potentially caused by genetic changes in the human leukocyte antigen (HLA), cause skin inflammation, and are characterized by abscesses in the GI and sinus tracts. **OBJECTIVE:** We sought to determine the extent of coincident IBD and HS and the associated risk factors. **METHODS:** The Ovid Medline database was searched for all current literature on the correlations between HS and IBD. Articles were then included and removed according to specific inclusion and exclusion criteria, and a systematic review was performed on the remaining articles. **RESULTS:** Thirteen papers that met the inclusion and the exclusion criteria were selected, and after data collection, a significant correlation was found between the development of HS and IBD. Most of the articles reviewed stated that the corresponding association was a causal link between the two diseases. Furthermore, there were various risk factors and comorbidities associated with HS and the development of IBD, including smoking, obesity, perianal disease, and genetic predispositions such as HLA-B27 mutations. **DISCUSSION:** Studies show that there is a potential correlation with HS and IBDs. Additional research should to determine the genetic correlations between HS and IBDs and the underlying pathophysiological mechanism. **KEYWORDS:** Hidradenitis suppurativa, irritable bowel diseases, dermatology, gastroenterology, autoimmunity

Hidradenitis suppurativa (HS) is a chronic inflammatory skin condition characterized by painful nodules, abscesses, and sinus tract formation, primarily affecting intertriginous areas. Although its exact etiology remains unknown, it is likely multifactorial and thought to involve follicular occlusion and inflammation likely influenced by genetic and environmental factors.[1] This occlusion causes follicles to swell, eventually leading to rupture and thus triggering an intense inflammatory response. Additionally, certain bacteria, such as *Staphylococcus* and *Streptococcus* species, are more abundant in HS lesions while protective species may be less prevalent compared to healthy individuals.[1] Finally, HS lesions exhibit upregulation of innate immune pathways, with elevated levels of inflammatory cytokines such as tumor necrosis factor (TNF)-α, interleukin (IL)-1, and IL-17.[1]

Previous studies have shown a correlation between HS and the *HLA-B27* gene mutation, suggesting a susceptibility to HS among individuals with this genetic predisposition.[2] Environmental factors, such as obesity, smoking, and sex, have also been implicated in the development of HS.[3] Obesity increases skin friction, thereby fostering the conditions conducive to HS development.[3] Moreover, hormonal changes and metabolic syndrome associated with obesity have been linked to an elevated risk of HS.[3] Nicotine, a component of cigarettes, is also known to exacerbate HS likely through increased follicular occlusion and slow healing.[3] Lastly, sex differences reveal that women are disproportionately affected by HS than men, with a ratio of 3:1.

In contrast, irritable bowel diseases (IBDs), encompassing Crohn's disease (CD) and ulcerative colitis (UC), are inflammatory gastrointestinal conditions characterized by diarrhea, abdominal pain, and bloody stools.[4] Like HS, the exact etiology of IBDs remain unknown, though both genetic and environmental factors are implicated. Studies have demonstrated a correlation between the *HLA-B27* gene mutation and the presence of IBDs.[5] Smoking has been identified as a risk factor for IBDs, particularly CD attributed to the proinflammatory effects of nicotine.[6] Obesity also exhibits strong associations with IBDs, affecting between 15 to 40 percent of IBD patients.[7] Additionally, dysbiosis, an imbalance in the gut microbiome, has emerged as a critical factor in IBD pathogenesis, contributing to immune dysregulation and chronic intestinal inflammation. Certain bacterial species, such as *Escherichia coli* and *Fusobacterium*, are often overrepresented in IBD patients, while protective species may be diminished, further exacerbating disease progression.[8] Sex differences also play a role in the manifestation of IBDs, with women being more predisposed to developing CD and men to UC.[9]

Despite the similarities between HS and IBDs in terms of inflammatory

FUNDING: No funding was provided for this article.
DISCLOSURES: The authors declare no conflicts of interest relevant to the content of this article.
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nature and the involvement of genetic and environmental factors, limited research has been conducted on the association between these two conditions. Nevertheless, existing studies suggest a potential link between HS and IBDs.[10] In this systematic review, we aim to synthesize current literature on the association between HS and IBDs and to evaluate the interplay between these conditions and whether individuals affected by one condition face an increased risk of developing the other.

METHODS

A systematic review was conducted on June 29, 2023, using three databases: Ovid Medline, PubMed, and the Cochrane Database. While all three databases were searched, Ovid Medline yielded the most results, with PubMed and Cochrane contributing minimal studies. The search terms included a combination of Medical Subject Headings (MeSH) and free-text terms, systematically combined using Boolean operators (AND, OR). Terms such as "Hidradenitis Suppurativa," "HS," "Inflammatory Bowel Disease," "IBD," "Crohn's Disease," "Ulcerative Colitis," and variations of these combinations (eg, "HS AND IBD" or "Hidradenitis Suppurativa AND Crohn's Disease") were used. Filters were applied to limit results to English-language articles and studies conducted in the United States.

The search initially resulted in 48 articles, with one duplicate removed, resulting in 47 eligible articles for screening. Titles and abstracts were then independently screened by two reviewers to assess relevance, and 22 articles were excluded for focusing on either HS or IBD independently without investigating their association. The remaining 25 full-text articles were sought for retrieval, though one article could not be accessed. Of the 24 articles retrieved, one was excluded for not being conducted in the United States, two were excluded for not being written in English, two discussed both HS and IBD but not in relation to each other, and seven were excluded for being systematic reviews or meta-analyses rather than original research studies. This resulted in a final selection of 12 articles that met both the inclusion and exclusion criteria and were included in the analysis.

Screening and selection were performed independently by two reviewers, with discrepancies resolved through discussion. The study adhered to Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines and followed principles from the Cochrane Handbook for Systematic Reviews of Interventions where appropriate. A formal risk of bias assessment was not conducted as this systematic review aimed to synthesize and summarize findings from the selected studies rather than perform a meta-analysis. Future meta-analyses examining the association between HS and IBD may benefit from applying standardized tools such as the Newcastle-Ottawa Scale (NOS) or ROBINS-I tool to systematically evaluate study quality.

Key data, including study design, population characteristics, prevalence rates, risk factors, findings, and limitations, were extracted from each included study and synthesized into Table 1 to provide a structured overview of the key findings. Figure 1 shows a visual representation of the paper and data collection discussed here, using the PRISMA model.

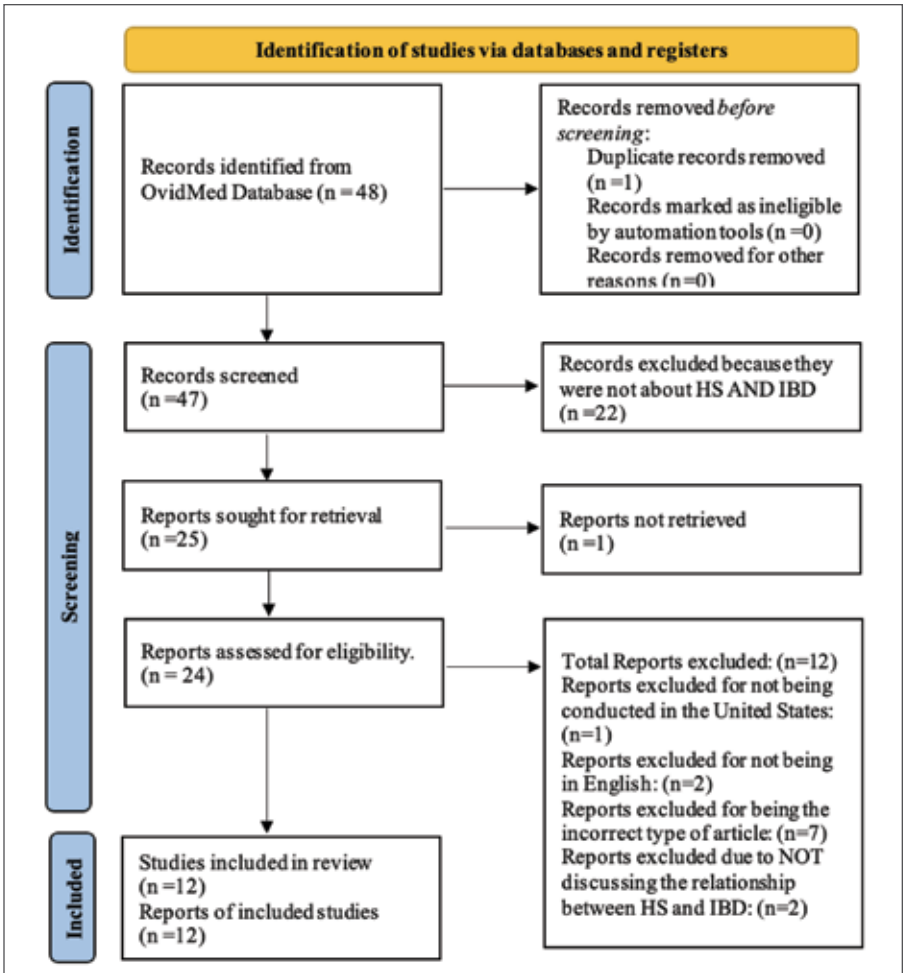

FIGURE 1. Flow chart on the methods of literature search

RESULTS

A total of 12 papers were included in the systematic review that fit the inclusion criteria. The study designs of these publications include cohort studies, cross-sectional studies, case-control studies, randomized controlled trials, bidirectional Mendelian randomization studies, and case reports. All studies discussed varying associations between HS and IBDs and conducted extensive data analysis regarding the two autoimmune conditions. A summary of these studies, including study design, population characteristics, prevalence rates, risk factors, key findings, and limitations is provided in Table 1.

The temporal relationships and associations between cutaneous manifestations and inflammatory bowel disease: a nationwide population-based

TABLE 1. Summary of the studies investigating the association between HS and IBD

REFERENCE	STUDY DESIGN	POPULATION	PREVALENCE/RISK OF HS AND IBD	RISK FACTORS	KEY FINDINGS/ CONCLUSIONS	LIMITATIONS
Hung et al[11]	Observational study	2,847 IBD patients, 14,235 controls	HS odds: 4% in IBD vs. 15% in controls	Presence of IBD, inflammatory pathways	HS incidence higher in IBD group	Observational study limitations
Ramos-Rodriguez[12]	Observational study	123,614 with atopic dermatitis, 6,806 with HS, 2,376,120 controls	HS increased risk of UC (HR 2.30) and CD (HR 2.70)	Underlying inflammatory pathways	Significant IBD risk in HS and psoriasis patients	Focus on in-hospital data
Janse et al[13]	Questionnaire-based study	1,260 patients (634 CD, 626 UC)	15% in CD, 6% in UC	Smoking, BMI, active IBD, perianal disease	Early onset HS associated with IBD	Small sample size, genetic focus
Yadav et al[14]	Longitudinal cohort study	679 IBD patients followed over 19.8 years	IBD pts 9x more likely to develop HS	Female gender, obesity	9x higher HS risk in IBD	Small HS sample size
Van Der Zee et al[15]	Cross-sectional study	158 IBD patients (102 CD, 56 UC)	16% prevalence of HS in IBD patients	Crohn's disease higher prevalence	Potential underreporting due to self-reports	Small sample size, reliance on self-reports
Tandon et al[16]	Case-control study	IBD patients at Mount Sinai Hospital	Active smokers (OR 10.3), past smokers (OR 8.4), perianal disease (OR 21.1)	Smoking, active endoscopic disease, perianal disease	Stronger association with smoking and perianal disease	Small sample, retrospective study
Deckers et al[17]	Cross-sectional study	1,076 HS patients	IBD prevalence: 3.3% (CD: 2.5%, UC: 0.8%)	Less obesity in HS+IBD	Higher IBD prevalence in HS patients	Potential underestimation of IBD prevalence
Neri et al[18]	Nested case-control study	125 HS patients (25 with IBD, 100 controls)	IBD less frequent in familial HS, lower obesity prevalence	Reduced familial HS, lower obesity rates	HS less severe in IBD group	Retrospective design, single study center
Bao et al[19]	Two-sample Mendelian randomization study	12,882 IBD patients, 21,770 controls	IBD increases HS risk (OR 1.34), UC (OR 1.27), CD (OR 1.18)	Genetic predispositions (UC, CD risks)	IBD increases HS risk; HS does not cause IBD	Reliance on GWAS data
Ross et al[20]	Longitudinal cohort study	627 HS patients	16% had at least one autoimmune disease	Autoantibodies (ANA positivity)	Higher autoimmune disease prevalence in HS	Retrospective design
Tandon et al[21]	Case-control study	38 HS+IBD patients, 136 controls	6x more likely to smoke, 11x more likely to be obese	Smoking, obesity	Strong smoking and obesity associations	Small sample size
Bettoli et al[22]	Case study	One 36-year-old woman	UC first, then HS, then PG	IBD likely triggered HS and PG	IBD onset may trigger HS and PG	Single patient case study

ANA: antinuclear antibody; BMI: Body Mass Index; CD: Crohn's disease; GWAS: genome-wide association studies; HR: hazard ratio; HS: hidradenitis suppurativa; IBD: irritable bowel diseases; OR: odds ratio; PG: pyoderma gangrenosum; UC: ulcerative colitis

cohort study.[11] Out of 2,847 patients with IBD and 14,235 controls, a study by Hung et al[11] found the odds ratio for HS to be 4 percent for patients with IBD (n=2,857) and 15 percent in patients without IBD (n=14,235). The study found that new diagnoses of HS, pyoderma gangrenosum (PG), erythema nodosum (EN), polyarteritis nodosa (PAN), psoriasis, rosacea, and aphthous stomatitis were significantly higher in IBD patients compared with controls. There was also a statistically significant increase in the cumulative probability of developing certain dermatological diseases in the IBD group. The incidence for dermatologic disease after diagnosis for IBD was 4.32 for the IBD group and 1.71 for the non-IBD group. Additionally, there were nine cases in the IBD group and 18 cases in the non-IBD group. The median time to develop a cutaneous manifestation after the diagnosis of IBD ranged from 3.08 years for cutaneous T cell lymphoma to 5.67 years for PG.[11]

The in-hospital burden of hidradenitis suppurativa in patients with inflammatory bowel disease: a decade nationwide analysis from 2004 to 2014.[12] A decade-long nationwide analysis by Ramos-Rodriguez et al[12] examined five major skin conditions: atopic dermatitis (123,614 patients), psoriasis (83,049 patients), alopecia areata (18,135 patients), vitiligo (9,003 patients), and HS (6,806 patients). The study also analyzed a control group of 2,376,120 individuals without these skin conditions. Over a median follow-up of 718 days, after adjusting for IBD risk factors, they found that patients with HS had a significantly higher risk of both UC (HR 2.30) and CD (HR 2.70). Patients with psoriasis had an increased risk of CD (HR 1.23) but not UC (HR 1.01). No significant increase in IBD risk was observed in patients with atopic dermatitis, alopecia areata, or vitiligo. These findings shed light on the complex relationships between these skin conditions and the risk of developing inflammatory bowel disease.[12]

Identification of clinical and genetic parameters associated with hidradenitis suppurativa in inflammatory bowel disease.[13] In a survey-based study by Janse et al,[13] out of 1,260 patients who replied to the questionnaire accurately, 634 had CD and 626 had UC. HS was present in 96 (15%) patients with CD and 38 (6%) patients with UC.[13] Results revealed that 6.8 to 10.6 percent

versus 1 to 2 percent of patients with HS had IBD. The prevalence of women in IBD with HS was significantly higher than in IBD without HS ($p<0.001$). Additionally, patients with HS were significantly younger than those without HS at 41.8 years compared to 47.9 years ($p<0.001$). Other independent associated parameters for HS include smoking and a higher body mass index. Comorbid HS is associated with early onset IBD in which anti TNF-α therapy and surgical resection are often needed. Suggestive protective association with *ELOVL7* (*P*=7.15 x10, odds ratio=0.4) and suggestive risk associated with genes *SULT1B1* and *SULT1E1* for HS in the context of IBD (*rs* number 201477 *P*=7.48 x10, odds ratio 2.3). Active IBD, perianal disease and smoking may be associated with HS in IBD.

Hidradenitis suppurativa in patients with inflammatory bowel disease: A population-based cohort study in Olmsted County, Minnesota.[14] IBD patients were nine times more likely to develop HS than the general population, with female predisposition. Six hundred seventy-nine patients followed up over a median of 19.8 years, eight patients with HS (mean age 44.4 +/- 8.3 years; seven women; six with obesity) incidence rate ratio of HS in IBD was 8.9 (95% CI, 3.6-17.5), 10 to 30-year cumulative incidence of HS was 0.85 percent and 1.55 percent, respectively. The prevalence of IBD in HS patients (3.3%) is 4 to 8 times higher than the prevalence in the general northern European population (0.41%–0.74%), however HS-IBD patients do not have a distinct HS phenotype.

Hidradenitis suppurativa and inflammatory bowel disease: Are they associated? Results of a pilot study.[15] This study was done to determine the prevalence of HS in patients with IBD living in the Southwest of Netherlands. Of the 158 patients interviewed attending a patient information meeting in Rotterdam in 2008, 102 (65%) had CD and 56 (35%) had UC. The patients were then asked if they experienced any painful boils in the axillae and/or groin over the course of their life, using two color pictures of HS at different stages to clarify the disease in question. Twenty-five (16%) reported having these boils at some point in their life, of whom 17 were patients with CD (17%) and eight had UC (14%). The study limitations included a small sample size of self-reported CD/UC patients in the southwest region of the Netherlands, as well as with self-reported boils that were not examined to confirm diagnosis.

Risk factors for hidradenitis suppurativa in patients with inflammatory bowel disease.[16] This study sought to determine the characteristics and risk factors for the development of HS in a cohort of patients with IBD at the Mount Sinai Hospital in May 2019. The researchers incorporated patients with IBD who developed HS and matched the cases 5:1 by age, gender, and IBD type (UC or CD) as controls for those with IBD and those without. The statistical method, logistic regression was used to calculate odd ratios (ORs) with 95 percent confidence intervals. Among the 29 patients who had HS, the severity was mild in 10 patients, moderate in 16 patients, and severe in three patients. Additionally, 37.9 percent (n=11) were male and 62.1 percent (n=18) were female. The researchers found that patients with HS and IBD were more likely to be active (OR 10.3, 95% CI 2.0 to 54.0, *P*=0.006) or past (OR 8.4, 95% CI 2.7 to 25.8, $p<0.005$) smokers. Patients with HS and IBD were also more likely to have active endoscopic disease (OR 3.8, 95% CI 1.2 to 12.2, *P*=0.022). Furthermore, those with HS and CD were more likely to have active perianal disease (OR 21.1, 95% CI 6.2 TO 71.9, $P<0.005$). Despite establishing that perianal disease, smoking and active IBD can be associated with HS in IBD. Larger studies are still needed to characterize this morbid condition.

Inflammatory bowel disease is associated with hidradenitis suppurativa: results from a multicenter cross-sectional study.[17] In this study, Deckers et al[17] sought to determine the prevalence of IBD in patients with HS by specifically determining whether those patients have a distinct phenotype. Commonly, HS is associated with IBD, but its prevalence in patients with HS has not been fully explored, necessitating this study. In 1,076 patients that were involved in the study, the researchers found that IBD has a prevalence rate of 3.3 percent (95% CI 2.3–4.4). The prevalence of CD was 2.5 percent (95% CI 1.6–3.4) and the prevalence of UC was 0.8 percent (95% CI 0.3–1.4). Patients with HS-IBD less frequently had obesity (13.9% vs. 31.2%, *P*=0.04) than patients with only HS, but there were no differences in gender, family history of HS, disease severity, body areas affected by HS, or smoking status. Generally, the study found that IBD occurs in 3.3 percent of patients with HS, a prevalence rate that is 4 to 8 times higher than the general population rate of 0.41 percent to 0.74 percent in Northern Europe. Patients with both HS and IBD were less likely to have obesity compared to those with HS only, but no distinct HS phenotype was observed. Nonetheless, the study acknowledged the possibility of underestimating IBD prevalence among HS patients, as some may develop IBD later.

Hidradenitis suppurativa and inflammatory bowel disease in a nested case-control study.[18] Neri et al conducted this study to establish the characteristics of HS in patients with IBD with or without concomitant IBD. The researchers employed a nested case-control study approach, where each patient with concomitant HS, having been classified according to the Hurley score and the International Hidradenitis Suppurativa Severity Score System (IHS4) was retrospectively matched with four patients with HS and no IBD controls for gender and age. From a population of 125 patients with HS, 25 of whom had IBD (76% Crohn's disease and 24% ulcerative colitis), and 100 matched controls with no IBD, the researchers found the following. Familial HS, obesity, and perianal HS were less frequent in cases than in controls (1 [4%] vs. 25 [25%]: $p=0.02$; 1[4%] vs. 21 [21]: $p=0.04$; 1 [4%] vs. 31 [31%]; $p=0.005$ respectively). HS was less severe in cases when assessed by the IHS4 (5.9 ± 4 vs. 9 ± 6.7; $p=0.04$). As for complete drug-induced response, it was more frequent in IBD (13 [53%] vs. 28 [28%]; $p=0.04$). In conclusion, the clinical characteristics of HS and of patients differed between cases and controls. Present findings suggest the need to appropriately search and assess skin lesions that are compatible with HS in IBD.

Causal association between inflammatory bowel disease and hidradenitis suppurativa: A two-sample bidirectional Mendelian randomization study.[19] A two-sample MR was performed using an analysis of 12,882 patients with 21,770 controls with IBD and its main subtypes, UC and CD.[19] A total of 409 cases and 211,139 controls without HS were included in the data for this condition from various genome-wide association studies. The researchers used ORs with 95 percent confidence intervals (CIs) to estimate causal effects. The causal relationship between HS and IBD was assessed in both

directions. The risk of HS was increased by IBD (IVW OR=134, 95% CI=1.20 –1.49, p=2.15E-07) and, in addition, HS was affected by UC (IVW OR=1.27, 95% CI=1.13-1.43, p=8.97E-04) and CD (IVW OR=1.18, 95% CI=1.08–1.29, p=4.15E-04). However, the study did not identify any causal relationship between HS and IBD or its subtypes (IBD IVW OR=1.00, 95% CI=0.96–1.05, p=0.85; UC IVW OR=0.99, 95% CI=0.95–1.03, p=0.65; CD IVW OR=1.03, 95% CI=0.98–1.07, p=0.28). As such, the study demonstrated that HS has no causal effect on IBD, while IBD and its subtypes, do have a significant causal effect on HS. The study emphasizes on further research to determine the pathophysiology of the causal relationship between IBD and HS including conducting gut-skin interactions.

Association of hidradenitis suppurativa with autoimmune disease and autoantibodies.[20] Ross et al conducted this longitudinal cohort study to determine whether there is a greater correlation between autoantibody specificities or autoimmune diseases and HS than there is between other specificities and HS severity. To achieve this objective, the researchers employed the SlicerDicer search tool to identify 627 patients, through a search criterion of at least one visit to the dermatology department and HS diagnosis by ICD-10 code L73.2. Charts were also reviewed to determine HS disease severity, treatment modalities, and presence of autoimmune disease and autoantibody positivity. Sixteen percent (n=101) of patients had at least one autoimmune disease, mostly in the form of thyroid disease, psoriasis, IBD, and lupus. Two hundred and twelve patients were tested for the presence of autoantibodies and 28.4 percent (n=54) were found to have antinuclear antibodies. Fifty-four patients with more severe HS disease manifestation required biological medications to treat their HS. Neither HS severity nor biologic treatment was associated with presence of autoimmune disease or positive autoantibodies. The researchers concluded that in this large cohort of patients with HS, the presence of autoantibodies was more prevalent than in normal populations.

Risk factors for developing hidradenitis suppurativa in patients with inflammatory bowel disease: A retrospective case-control study.[21] Following small studies and case reports suggesting the possible association between HS and IBD, Lukach et al[16] performed a case-control study to further characterize IBD patients with HS in terms of body mass index, smoking status, sites affected HS, IBD medication history, and IBD type and features.[21] They identified 38 patients with HS and IBD and matched them based on gender, age, and IBD type to 136 controls with IBD but not HS. Among the patients with HS and IBD, the most common sites were the inguinal, axillary and perianal regions. Compared to IBD patients alone, those with HS and IBD were almost six times as likely to be current smokers (p<0.01) and nearly 11 times more likely to have obesity (p<0.01). Patients with CD and HS are significantly more likely to have perianal and ileocolonic disease than patients with CD only (OR 831, 95% CI 2.90–23.80 and OR 2.85, 95% CI 1.19–6.81, respectively; p<0.01 for both). In summary, compared to patients with IBD who never develop HS, patients who have both IBD and HS are more likely have obesity or be overweight, to be current or former smokers, and to have ileocolonic or perianal disease.

The association among pyoderma gangrenosum, ulcerative colitis, and hidradenitis suppurativa and the syndromic hidradenitis suppurativa network: A case report.[22] Bettoli et al observed the concurrent occurrence of HS, PG, and IBD UC, leading to them describing a new association. The case report involved a 36-year-old woman who first presented with IBD, followed shortly by the development of HS and PG. She was referred due to a rapid and severe worsening of both gastrointestinal and systemic symptoms, ultimately requiring a total colectomy. A severe aggravation of both GI symptoms and general systemic situation led to total colectomy. Shortly after the surgical treatment of UC, the cutaneous manifestations of PG and HS with no specific treatment disappeared almost completely, presenting the possibility of a common etiopathogenetic mechanism and possibly an inductor role of UC on the other disorders. The description of this association suggests that treatment-induced resolution of one of the associated conditions could lead to the spontaneous clearance of one or more of the others. It suggests the possible presence of a pathogenic link between them and the pivotal role of one of them, which in this case was colitis.

DISCUSSION

The studies reviewed here have extensively explored the intricate connection between two chronic inflammatory conditions, HS and IBD. While diverse causative factors are identified across these studies, they collectively suggest that both conditions likely stem from a combination of genetic predisposition and environmental influences.

Previous studies have shown significant association between HS and IBD. One published abstract found that individuals with IBD have an increased prevalence of HS, around 3 percent, compared to the general population.[23] It also reported that those with Crohn's disease have a higher risk of HS compared to those with UC.[23] Genetic factors have also been theorized to play a role in the association, specifically mutations in *HLA-B27* gene.[1,2] Furthermore, environmental factors, such as obesity and smoking, increase the association between HS and IBD.[3,6,13,21] Obesity, for example, not only fosters the development of HS through increased skin friction, but also correlates with metabolic syndrome, which is a shared risk factor for both HS and IBD.[3,6,13,21] In addition, research suggests that females are more likely to have HS,[24] while males with IBD are more likely to develop HS.[23] However, only one study among those reviewed identified a potential hormonal link between HS and IBD, noting that females were more likely to develop HS.[14] Further research is required to clarify the role of hormonal factors in the pathophysiology of these conditions.

Sex disparity is also a notable factor, as women show a higher prevalence of HS.[24] The incidence of IBD also differs by gender depending on the subtype, with women having a greater predisposition to Crohn's disease compared to ulcerative colitis.[25] In this study, most of the reviewed studies did not explicitly identify a hormonal link between HS and IBD, physiological and metabolic factors, potentially influenced by hormonal differences, may contribute to this sex-specific susceptibility. Moreover, the emerging concept of the gut-skin axis, elucidating the intricate communication between the gut microbiome and the skin, holds promise in comprehending this association.

However, numerous inquiries remain unanswered. Future research endeavors should prioritize unraveling the underlying mechanisms driving the co-occurrence of IBD and HS. Investigating the potential influence of the gut

microbiome on HS development and delving into the specific genetic and immunological factors implicated therein represent promising avenues for exploration.

CONCLUSION

The studies reviewed herein provide substantial evidence for the association between IBD and HS. They reveal the importance of holistically diagnosing and treating patients with IBD and HS, including addressing the gastrointestinal and cutaneous underpinnings of these conditions.

Nonetheless, the field remains open for research and further analysis given that understanding the shared immunological and genetic bases, exploring the connection between the gut and skin, and investigating treatment procedures could provide new insights on how to treat this condition and further enhance patient lives.

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