Journal of the Canadian Association of Gastroenterology, 2021, 4(4), 165–172
doi: 10.1093/jcag/gwaa024
Original Article
Advance Access publication 28 July 2020

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Risk Factors for Developing Hidradenitis Suppurativa in Patients With Inflammatory Bowel Disease: A Retrospective Case–Control Study

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Abstract

Background: Hidradenitis suppurativa (HS) is associated with inflammatory bowel disease (IBD), though risk factors remain to be determined.
Aim: To characterize HS among a cohort of IBD patients and identify risk factors for its development.
Methods: This was a retrospective case–control study at the ambulatory IBD centre at Mount Sinai Hospital from inception to May 2019. Patients with IBD who developed HS were included. Cases were matched 5:1 by age, gender (male versus female) and IBD type (ulcerative colitis [UC] or Crohn's disease [CD]) to controls who had IBD without HS. Conditional logistic regression was used to calculate odds ratios (ORs) with 95% confidence intervals (95% CIs).
Results: Twenty-nine cases of HS (19 CD and 10 UC) and 145 controls were included. Of the 29 patients with HS, 11 (37.9%) were male and 18 (62.1%) were female. The severity of HS was mild in 10 (34.5%), moderate in 16 (55.2%) and severe in 3 (10.3%) patients. 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, $P < 0.005$).
Conclusions: Active IBD, perianal disease and smoking may be associated with HS in IBD. Larger studies are needed to better characterize this morbid condition.

Keywords: *Hidradenitis; IBD; Perianal; Skin rash*

INTRODUCTION

Inflammatory bowel disease (IBD) is a chronic relapsing inflammatory condition that includes Crohn's disease (CD) and ulcerative colitis (UC). Almost a third of patients with IBD develop extraintestinal manifestations (EIMs) (1), including musculoskeletal, ophthalmologic and hepatobiliary involvement (2,3). Furthermore, numerous mucocutaneous manifestations of IBD have been described such as erythema nodosum, pyoderma gangrenosum (PG) and aphthous stomatitis (3).

More recently, an association between IBD and hidradenitis suppurativa (HS) has been reported (4–7). HS is a chronic inflammatory follicular skin disease characterized by painful abscesses and sinus tracts in areas rich of apocrine glands such as the axillae, inguinal and anogenital regions. It is associated with significant morbidity and reduction in quality of life (8) and in rare cases, the development of squamous cell carcinoma (9). Common immunological, hereditary and microbiome pathways may predispose patients with IBD to the development of HS. In fact, the risk of HS among patients with IBD appears to be

ninefold higher as compared to the risk in the general population, with a reported incidence of 0.55 cases per 1000 person-years (4,6,7). Similar to IBD, HS can also be associated with inflammatory conditions including spondyloarthropathies and is occasionally managed with biologic therapy (10). Active smoking (11) and obesity (4) appear to be strong risk factors for the development of HS in IBD. Furthermore, HS appears more commonly seen in patients with CD compared to UC, particularly in those with active fistulizing or perianal disease (11).

While the association between HS and IBD has been described, there is a need for more studies better characterizing the link between these two conditions. As such, we aimed to describe the clinical characteristics of the dermatologic features of HS in patients with IBD as well as IBD-specific features in these patients. Furthermore, we aimed to determine risk factors for the development of HS among patients with IBD, with a particular emphasis on disease activity.

METHODS

This was a retrospective case–control study performed at Mount Sinai Hospital, University of Toronto. It was approved by the research ethics board at Mount Sinai Hospital (MSH REB 18-0283-C).

Patient Selection and Data Collection

All ambulatory patients, aged >18 years of age, with UC, CD or indeterminate colitis followed in the clinics of the Mount Sinai Hospital IBD Centre, from inception to May 2019, were identified for chart review. Those with HS as diagnosed by the primary gastroenterologist or dermatologist on physical examination were included as cases in this study. In particular, patients with HS were identified by searching the electronic medical record for key terms such as 'Hidradenitis Supprativa', 'Hidradenitis', 'suppurativa', 'skin condition' or 'skin boils'. These cases were matched 5:1 to controls with IBD who did not develop HS by age at IBD diagnosis, gender (male versus female) and IBD subtype (UC versus CD).

The electronic medical record was searched retrospectively for the collection of the following data: (a) baseline demographics including age, gender, most recent body weight or that at time of HS diagnosis, smoking status (no smoking history, past smoking history, active smoking at time of HS diagnosis), alcohol intake (reported by the patient on history and defined as any amount of alcohol intake at time of HS diagnosis) and other comorbidities (including respiratory diseases, such as chronic obstructive pulmonary disease, reactive airway disease or interstitial lung disease, cardiac diseases, such as ischemic disease or congestive heart failure, and any mental health disorder); (b) IBD characteristics including IBD subtype (UC versus CD versus indeterminate colitis), age at IBD diagnosis, current IBD-related medications at time of HS diagnosis, disease location, disease phenotype and disease activity (endoscopic, clinical or both) and (c) HS characteristics including timing of HS diagnosis (before IBD diagnosis, at time of IBD diagnosis, after an IBD diagnosis), location (axillae, inguinal, extremities, perianal or other), severity stage as defined by the Hurley system (stage 1: abscess formation without sinus tracts and scarring; stage 2: recurrent abscesses with sinus tracts; stage 3: diffuse interconnected sinus tracts and abscesses) (12) and HS-related treatment (antibiotics, local steroid injection, surgical excision or other modality). Finally, a dermatology consultation report, specifically addressing HS, was searched for in the medical record and additional data elements were collected if available.

Outcomes and Definitions

The primary outcome of this study was to better characterize the clinical characteristics of the dermatologic features of HS in patients with IBD as well as IBD-specific features in these patients. Risk factors for the development of HS in IBD were explored, with a particular emphasis on disease activity. Clinical disease activity was defined as a modified Harvey-Bradshaw Index (HBI) score >5 or partial Mayo (pMayo) score >2. For consistency, clinical disease activity was obtained either 3 months before or 3 months after the diagnosis of HS. Endoscopic disease activity was defined as the presence of any active inflammation (identified by the index endoscopist) on sigmoidoscopy or ileocolonoscopy within a calendar year before or after diagnosis of HS. For patients without HS, clinical and endoscopic disease activity was recorded in the same age year as the matched cases. Finally, the severity of HS was stratified by the aforementioned Hurley classification (12).

Statistical Analysis

Risk factors were defined as dichotomous values for statistical purposes. Categorical comparisons between cases and controls were analysed using the Pearson chi-squared test (if sample size was ≥10) or Fisher's exact test (if sample size was <10). Median values with interquartile ranges (IQRs) were calculated for variables such as age, weight, disease activity scores and anti-tumour necrosis factor (TNF) drug levels. The Mann–Whitney *U*-test was used to calculate differences in these variables between patients who developed HS compared to matched controls. To determine specific associations with the development of HS in IBD, odds ratios (ORs) with 95% confidence intervals (95% CIs) were calculated using conditional binomial logistic regression analysis. A P value <0.05 was considered statistically significant. All analyses were performed using the SPSS software version 26 (Chicago, IL).

RESULTS

A total of 29 patients with IBD were diagnosed with HS and were matched to 145 patients with IBD without HS (Table 1). Of the 29 patients with IBD and HS, 11 (37.9%) were male and

18 (62.1%) were female. Ten (34.4%) were diagnosed with UC and 19 (65.6%) were diagnosed with CD. The median weight of patients with IBD and HS was 82.0 kg (IQR 41.5 kg) compared to 65.1 kg (IQR 21.5 kg) for those without HS ($P = 0.008$). Compared to those without HS, patients with IBD and HS were more likely to be current (4 of 29 [13.8%] versus 3 of 145 [2.1%]) or past (9 of 29 [31.0%] versus 9 of 145 [6.2%]) smokers ($P < 0.005$). Similarly, compared to those without HS, patients with IBD and HS were more likely to have any active alcohol intake (17 of 29 [60.0%] versus 43 of 145 [29.6%], $P = 0.003$) at time of HS diagnosis. There was no difference in the presence of other comorbidities between patients with IBD and HS compared to those without HS.

Characteristics of Hidradenitis Suppurativa

All 29 patients with HS were diagnosed with their skin condition after being diagnosed with IBD. All patients were seen by a dermatologist for their HS. The median age of HS diagnosis was 25 years (IQR 10 years). Of the 10 patients with UC who developed HS, the areas affected were axillae only in 5 (50.0%) patients, inguinal only in 2 (20.0%) patients and both axillae and inguinal in 3 (30.0%) patients (Table 2). Four (40%) patients were treated with antibiotics for the HS, one (10.0%) had local steroid injections and four (40.0%) had surgical excision of the HS. The severity of HS was mild (stage 1) in four (40.0%) patients, moderate (stage 2) in five (50.0%) of patients and severe (stage 3) in one (10.0%) patient. Of the 19 patients with CD who developed HS, the areas affected were axillae only 2 (10.5%) patients, inguinal only in 3 (15.8%) patients, extremity only in 1 (5.3%) patient, perianal only in 2 (10.5%) patients and both axillae and inguinal in 8 (42.1%) patients. Ten (52.6%) patients were treated with antibiotics for the HS, two (10.5%) had local steroid injections and four (21.0%) had surgical excision of the HS. The severity of HS was mild (stage 1) in 6 (31.6%) patients, moderate (stage 2) in 11 (57.9%) of patients and severe (stage 3) in 2 (10.5%) patients. Overall, there were no differences in characteristics of HS in patients with UC and CD (Table 2).

Inflammatory Bowel Disease Characteristics in Patients With and Without Hidradenitis Suppurativa

The median age of IBD diagnosis in patients with HS was 20 years (IQR 9 years) and in those without HS was 16 years (IQR 8 years) ($P = 0.098$). Disease characteristics of CD and UC were compared between patients who developed HS and those who did not (Table 3). Compared to those with CD without HS, those with CD and HS were more likely to have a fistulizing CD phenotype (12 of 19 [63.2%] versus 21 of 94 (22.3%), $P = 0.0003$), history of perianal disease (16 of 19 [84.2%] versus 35 of 94 [37.2%], $P < 0.0005$) and active perianal disease at time of HS diagnosis (16 of 19 [84.2%] versus 15 of 94 [16.0%], $P = 0.0005$). There was no difference in disease location (ileal versus ileocolonic versus colonic) or prior CD-related surgery between the two groups. Similarly, there was also no difference in UC disease location or history of UC-related surgery in patients who developed HS compared to those who did not.

Table 1. Baseline characteristics of patients with IBD at time of diagnosis with HS compared to those with IBD without HS

	Patients with HS, $N = 29$	Patients without HS, $N = 145$	P value
IBD diagnosis, median age (IQR)	20 (9)	16 (8)	0.098
Gender			N/A
Male	11 (37.9%)	55 (37.9%)	
Female	18 (62.1%)	90 (62.1%)	
Smoking status			**<0.005**
Current smoker	4 (13.8%)	3 (2.1%)	
Prior smoker	9 (31.0%)	9 (6.2%)	
No smoking history	15 (51.7%)	130 (89.6%)	
Unknown	1 (3.5%)	3 (2.1%)	
Alcohol intake history			**0.003**
Yes, current intake	17 (60%)	43 (29.6%)	
None or past history	12 (40%)	102 (70.4%)	
Weight (kg), median (IQR)	82.0 (41.5)	65.1 (21.5)	**0.008**
Other comorbidities			
Cardiac	2 (6.8%)	3 (2.1%)	0.194
Respiratory	2 (6.8%)	3 (2.1%)	0.194
Mental health	2 (6.8%)	10 (6.9%)	1.00

Values in bold are statistically significant. HS, Hidradenitis suppurativa; IBD, Inflammatory bowel disease; IQR, Interquartile range; N/A, not applicable.

Table 2. Characteristics of HS in patients with inflammatory bowel disease

	Patients with HS		
	Ulcerative colitis (10 patients)	Crohn's disease (19 patients)	*P* value
Age at HS diagnosis, median (IQR)	24.0 (10.0)	25.0 (12.0)	
Location			0.173
Axillae only	5 (50.0%)	2 (10.5%)	
Inguinal only	2 (20.0%)	3 (15.8%)	
Extremities	0 (0%)	1 (5.3%)	
Perianal	0 (0%)	2 (10.5%)	
Axillae inguinal	3 (30.0%)	8 (42.1%)	
Other	0 (0%)	3 (15.8%)	
Treatment			
Antibiotics	4 (40.0%)	10 (52.6%)	0.518
Local steroid	1 (10.0%)	2 (10.5%)	0.965
Surgical excision	4 (40.0%)	4 (21.0%)	0.278
Hurley stage			0.562
1 (mild)	4 (40.0%)	6 (31.6%)	
2 (moderate)	5 (50.0%)	11 (57.9%)	
3 (severe)	1 (10.0%)	2 (10.5%)	

HS, Hidradenitis suppurativa; IQR, Interquartile range.

Compared to patients without HS, those with HS were more likely to be managed with antibiotics (3 of 29 [10.3%] versus 1 of 145 [0.7%], $P = 0.015$) for fistulizing complications (Table 3). There was no difference in anti-TNF use between the two groups (16 of 29 [55.1%] versus 76 of 145 [52.4%], $P = 0.840$). Furthermore, there was no difference in median anti-TNF drug levels at time of HS diagnosis compared to those without HS for infliximab (7.0 μg/g [10 patients] versus 7.9 μg/g [44 patients], $P = 0.429$) and adalimumab (12.0 μg/g [3 patients] versus 12.9 μg/g [14 patients], $P = 0.950$).

Finally, the presence of EIMs was compared between patients who developed HS and those who did not (Table 3). Compared to those who did not develop HS, patients with IBD and HS were numerically more likely to have a history of PG (2 of 29 [6.8%] versus 1 of 145 [0.7%], $P = 0.072$). Otherwise, there were no other differences in presence of EIMs in patients with IBD with HS compared to those without HS.

Disease Activity and Risk of Hidradenitis Suppurativa

Compared to those without HS, patients with HS were more likely to have clinically active IBD within 6 months of HS diagnosis (14 of 29 [48.3%] versus 35 of 145 [24.1%], $P = 0.008$) (Table 3). Similarly, compared to those without HS, patients with HS were also more likely to have endoscopically active disease (18 of 29 [62.1%] versus 31 of 114 [27.2%], $P < 0.005$). Specifically, in patients with UC, the median partial Mayo score in patients with HS was 2 (IQR 5) compared to a median partial Mayo score of 0 (IQR 2) in those without HS ($P = 0.016$). The endoscopic Mayo score in patients with UC and HS was 2 (IQR 1), compared to an endoscopic Mayo score of 0 (IQR 1) in those with UC but no HS ($P = 0.031$). For patients with CD, those who developed HS had a median HBI score of 3.5 (IQR 8), whereas those who did not develop HS had a median HBI score of 1 (IQR 3) ($P = 0.008$).

Logistic Regression Analysis

On conditional binomial logistic regression analysis, patients with HS and IBD were more likely to have a past history of smoking (OR 8.4, 95% CI 2.7 to 25.8, $P < 0.005$) or were actively smoking at time of HS diagnosis (OR 10.3, 95% CI 2.0 to 54.0, $P = 0.006$) (Table 4). Similarly, these patients were more likely to have active IBD (a composite of clinical scores and endoscopic assessment) (OR 5.4, 95% CI 2.3 to 12.5, $P < 0.005$). This held true for active disease as defined by endoscopy (OR 3.8, 95% CI 1.2 to 12.2, $P = 0.022$) but not by clinical scores (OR 1.2, 95% CI 0.4 to 3.9, $P = 0.740$). Furthermore, patients with CD and HS were more likely to active perianal disease (OR 21.1, 95% CI 6.2 to 71.9, $P < 0.005$) but not stricturing (OR 1.1, 95% CI 0.3 to 4.6, $P = 0.899$) or fistulizing (OR 0.4, 95% CI 0.1 to 1.5, $P = 0.173$) disease. There were no differences in IBD-related therapies or extraintestinal manifestations between cases and controls.

DISCUSSION

The development of HS in IBD has been associated with significant morbidity. As such, determining specific risk factors for HS in this population may allow for early intervention which could

Table 3. Characteristics of inflammatory bowel disease in patients with and without HS

	Patients with HS	Patients without HS	*P* value
IBD patients	29 patients	145 patients	
IBD-related therapy			
Corticosteroids	4 (13.8%)	10 (6.9%)	0.256
5-ASA	5 (17.2%)	21 (14.4%)	0.775
Antibiotics	3 (10.3%)	1 (0.7%)	**0.015**
Methotrexate	5 (17.2%)	12 (8.3%)	0.312
Azathioprine	1 (3.4%)	13 (9.0%)	0.470
Anti-TNF	16 (55.1%)	72 (49.6%)	0.840
Infliximab	12 (75.0%)	52 (72.2%)	0.525
Adalimumab	4 (25.0%)	20 (27.8%)	1.000
Vedolizumab	2 (6.8%)	14 (9.7%)	0.502
Ustekinumab	3 (10.3%)	10 (6.9%)	0.444
Tofacitinib	0 (0%)	1 (0.7%)	0.833
Extraintestinal manifestations			
Rheumatoid arthritis	1 (3.4%)	2 (1.4%)	0.423
Erythema nodosum	3 (10.3%)	5 (3.4%)	0.132
Other arthritis	1 (3.4%)	2 (1.4%)	0.423
Pyoderma gangrenosum	2 (6.8%)	1 (0.7%)	0.072
Psoriasis	3 (10.3%)	5 (3.4%)	0.130
Other	1 (3.4%)	16 (11.0%)	0.312
Clinical disease activity[a]	14 (48.3%)	35 (24.1%)	**0.008**
Partial Mayo score	2 (IQR 5)	0 (IQR 2)	**0.016**
Harvey-Bradshaw Index	3.5 (IQR 8)	1 (IQR 3)	**0.008**
Endoscopic disease activity[b]	18 (62.0%)	31/114 (27.2%)	**<0.005**
Crohn's disease	19 patients	94 patients	
Location			0.135
Ileal	1 (5.3%)	19 (20.2%)	
Ileocolonic	15 (78.9%)	52 (55.3%)	
Colonic	3 (15.8%)	23 (24.4%)	
Type			
Stricturing	5 (26.3%)	30 (31.9%)	0.788
Fistulizing	12 (63.2%)	21 (22.3%)	**0.0003**
Hx of perianal	16 (84.2%)	35 (37.2%)	**<0.0005**
Active perianal	16 (84.2%)	15 (16.0%)	**0.0005**
History of ICR	2 (10.5%)	23 (24.5%)	0.236
History of ileostomy	0 (0%)	7 (7.4%)	0.351
Ulcerative colitis	10 patients	51 patients	
Location			0.159
Extensive colitis	5 (50.0%)	37 (72.5%)	
Left-sided colitis	3 (30.0%)	11 (21.6%)	
Proctosigmoiditis	2 (20.0%)	3 (5.8%)	
History of IPAA	0 (0%)	5 (9.8%)	0.580

Values in bold are statistically significant. 5-ASA, Aminosalicylate; HS, Hidradenitis suppurativa; IBD, Inflammatory bowel disease; ICR, Ileocecal resection; IPAA, Ileo-pouch anal anastomosis; TNF, Tumour necrosis factor.

[a]Clinically active disease was defined as a Harvey-Bradshaw Index score >5 or partial Mayo score >2.

[b]Endoscopic disease activity defined by the presence of erythema, friability or ulceration at time of sigmoidoscopy or colonoscopy.

Table 4. Logistic regression analysis for risk factors for the development of HS in IBD

	Odds ratio[a] (95% CI)	*P* value
Smoking status		
Never	1.0	N/A
Former	8.4 (2.7–25.8)	**<0.005**
Current	10.3 (2.0–54.0)	**0.006**
Current alcohol use	0.4 (0.2–1.2)	0.102
IBD activity		
Active clinical[b] or endoscopic[c] disease	5.4 (2.3–12.5)	**<0.005**
Active clinical disease[b]	1.2 (0.4–3.9)	0.740
Active endoscopic disease[c]	3.8 (1.2–12.2)	**0.022**
Crohn's disease		
Stricturing disease	1.1 (0.3–4.6)	0.899
Fistulizing disease	0.4 (0.1–1.5)	0.173
History of perianal disease	6.7 (2.1–21.8)	**0.001**
Active perianal disease	21.1 (6.2–71.9)	**<0.005**
History of ICR	0.4 (0.1–1.7)	0.197
Ulcerative colitis		
Pancolitis	4.9 (0.6–37.1)	0.121
Left-sided	2.4 (0.3–22.0)	0.426
IBD-related therapy		
Corticosteroids	0.5 (0.1–1.8)	0.267
5-ASA	0.6 (0.2–2.1)	0.389
Antibiotics	0.04 (0.01–0.5)	**0.011**
Methotrexate	0.3 (0.1–1.1)	0.079
Azathioprine	1.6 (0.2–13.7)	0.654
Anti-TNF agents	0.6 (0.2–1.5)	0.250
Vedolizumab	0.8 (0.2–3.6)	0.730
Ustekinumab	1.6 (0.4–6.3)	0.507
Extraintestinal manifestations		
Rheumatoid arthritis	0.9 (0.03–21.9)	0.959
Erythema nodosum	0.4 (0.06–3.3)	0.421
Other arthritis	0.6 (0.02–19.1)	0.747
Pyoderma gangrenosum	0.1 (0.004–2.6)	0.165
Psoriasis	0.3 (0.07–1.4)	0.123
Other	6.8 (0.4–105.6)	0.172

Values in bold are statistically significant. 5-ASA, Aminosalicylate; HS, Hidradenitis suppurativa; IBD, Inflammatory bowel disease; ICR, Ileocecal resection; IPAA, Ileo-pouch anal anastomosis; N/A, not applicable; TNF, Tumour necrosis factor.

[a]Matched for age, gender and type of IBD (ulcerative colitis vs. Crohn's disease).

[b]Clinically active disease was defined as a Harvey-Bradshaw Index score >5 or partial Mayo score >2.

[c]Endoscopic disease activity defined by the presence of erythema, friability or ulceration at time of sigmoidoscopy or colonoscopy.

lead to more effective treatment and improvement in overall quality of life. Previous studies have linked smoking and obesity as risk factors for the development of HS in patients with IBD (4,11). Here, we demonstrate that compared to those without HS, patients with HS and IBD are also more likely to have a smoking history, have active endoscopic IBD and have concurrent active perianal CD. Furthermore, there were no differences in overall HS characteristics in patients with UC and CD with respect to HS severity, location and overall management.

The association of HS in IBD appears to be stronger in patients with CD compared to those with UC. In fact, about two-third of the patients who developed HS in our study had CD, which is consistent with previous studies (7,13). Furthermore, fistulizing and perianal disease appear to be more common in patients with CD and HS compared to those who did not develop this skin condition. This may, however, be confounded by the fact that in routine practice, it may be quite challenging to distinguish perianal HS from fistulizing CD because of similar clinical presentation (11). In this setting, referral to a dermatologist and perianal imaging may assist in determining the correct diagnosis and appropriate management.

The association between CD and HS that has been demonstrated in the literature suggests common pathophysiological features between these two disease processes. Both CD and HS are characterized by the formation of sinus tracts and both share common risk factors such as smoking (14) and obesity (15). Moreover, common immunological pathways, such as dysregulation of interleukin-23/Th-17 and TNF-a, may be implicated in patients with both CD and HS (16). Previous studies have demonstrated that certain genes, such as *SULT1B1* and *SULT1E1* which are implicated in estrogen homeostasis, may be associated with the development of HS in CD (13). Finally, alterations in intestinal and skin microbiota may result in significant immune dysregulation, that is exacerbated by nicotine exposure, and an increased risk of HS in patients with IBD (17,18).

We also demonstrated that patients with IBD and HS were numerically more likely to have concurrent dermatological conditions such as PG, psoriasis and erythema nodosum compared to controls. Previous case reports and case series have suggested that patients with HS are at risk of developing PG (19,20). This also hints at common pathophysiologic underpinnings and a propensity towards developing dermatologic manifestations in a subset of IBD patients. As such, patients with IBD who develop HS or PG may benefit from close surveillance for the early detection of other potentially morbid skin conditions, particularly given their association with poor overall outcomes in IBD (21).

Consistent with previous studies, we also demonstrate that smoking and increased bodyweight may be associated with the development of HS in IBD (4, 11). In fact, Lukach et al. demonstrated that patients with HS and IBD were about 6

times more likely to have actively smoking at time of HS diagnosis and almost 11 times more likely to have concurrent diagnosis of obesity (11). Furthermore, compared to those who did not develop HS, we found that patients with IBD and HS were more likely to have active endoscopic disease. As such, close surveillance for dermatological diseases such as HS in patients with active IBD may allow for early identification and management of this morbid condition.

Interestingly however, despite this association of active disease with HS, we did not detect an increase in corticosteroid or biologic use in this cohort of patients. The literature is conflicting in the use of anti-TNF therapies in patients with IBD and HS. Whereas Janse et al. demonstrated that patients with HS and IBD were more likely to be prescribed anti-TNF therapy compared to those with IBD without HS (13), Lukach et al. did not find any significant difference between the two groups (11). These differences may be due to study design (questionnaire (13) versus case–control (11)) and patient characteristics. In our study, we included patients from a single tertiary care referral ambulatory clinic which may have enrolled patients with more aggressive disease phenotypes. This may have resulted in an increased use of anti-TNF therapy compared to the general gastroenterology practice in our study.

To our knowledge, our study is the first to correlate IBD activity with the development of HS. Given the relatively rare incidence of HS, we completed a retrospective case–control study to determine risk factors for the development of this morbid dermatological condition in IBD. Furthermore, all cases of HS were confirmed in consultation with a trained dermatologist at our university. However, there remains several inherent limitations. Our study was a single-centre study with a small number of patients diagnosed with HS. The small sample size might have resulted in type 2 error when assessing for associations between risk factors and development of HS in IBD. The retrospective nature of this study limited the completeness of available data and contributed to recall bias. For example, we were unable to determine whether the degree of severity of underlying IBD by endoscopic scores (i.e., simple endoscopic score for CD) and laboratory investigations (i.e., serum C-reactive protein or fecal calprotectin) is associated with an increased risk of HS. Similarly, variables such as the patient's height were not recorded in all charts and as such, a body mass index could not be calculated. Other factors that may increase the risk of HS, such as oral contraceptive use and family history, were also not routinely available on chart review. Though we attempted to control for disease activity by recording clinical parameters at the same time point for all patients, because of the nature of this study, this reflects a static time point. Disease activity is a dynamic process and our analysis may not truly reflect the underlying inflammation at the time of HS diagnosis. Furthermore, given the case–control study design, we are unable to report any 'cause-and-effect' conclusions and can only identify possible associations that require confirmation in future prospective studies. Finally, we were unable to determine the effect of HS on the quality of life of IBD patients and whether therapeutic intervention improved the HS and associated morbidity.

Overall, we demonstrate that compared to those without HS, patients with IBD and HS are more likely to have active endoscopic disease, and in cases of CD, active perianal disease. Furthermore, patients with HS and IBD may also have other concurrent dermatological conditions such as PG though this did not reach statistical significance. As such, appropriate counselling and identification of specific disease features may allow for early detection of HS and implementation of appropriate therapy. More studies better characterizing HS in IBD patients are needed to further elucidate the shared pathophysiology between these two potentially morbid chronic inflammatory conditions.

Funding

None.

Conflicts of Interest: P.T., V.G. and Z.G. have no disclosure of potential conflicts of interest. A.W. has served on the Advisory Board of Janssen, AbbVie, Takeda and Ferring. A.W. has served as a speaker for Janssen and AbbVie.

Author Contributions: P.T.: Study concept design, data acquisition, data analysis, drafting of manuscript and critical revision of manuscript. V.G.: Data analysis, drafting of manuscript and critical revision of manuscript. Z.G.: Data analysis and critical revision of manuscript. A.W.: Study concept design, data acquisition, data analysis, drafting of manuscript and critical revision of manuscript. All authors have approved the final version of the article submitted including the authorship list.

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