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Hidradenitis Suppurativa in Patients with Inflammatory Bowel Disease: A Population-Based Cohort Study in Olmsted County, Minnesota

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Abstract

Background and Aims—Patients with inflammatory bowel disease (IBD) may be at higher risk for hidradenitis suppurativa (HS). We studied the risk and clinical characteristics of HS in a population-based cohort of patients with IBD.

Methods—We identified all cases of HS (confirmed by biopsy and/or dermatologic evaluation) in a population-based inception cohort of Olmsted County, Minnesota, residents diagnosed with IBD between 1970 and 2004 and followed through August 2013. We estimated the incidence rate ratio (IRR) of HS in patients with IBD compared to the general population, and described the clinical characteristics, risk factors, and management of HS.

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Results—In 679 IBD patients followed over a median of 19.8 years, we identified 8 patients with HS (mean age, 44.4±8.3 years; 7 females; 6 obese). Compared to the general population, the IRR of HS in IBD was 8.9 (95% CI, 3.6–17.5). The 10- and 30-year cumulative incidence of HS was 0.85% and 1.55%, respectively. Five patients had Crohn's disease, four of whom had perianal disease; of 3 patients with ulcerative colitis, 2 had undergone ileal-pouch anal anastomosis. Axillae, groin and thighs were the most common sites of involvement. Six patients had Hurley stage 2 disease (recurrent abscesses with sinus tracts and scarring, involving widely separated areas), and required a combination of antibiotics and surgery; none of the patients were treated with anti-tumor necrosis factor- α agents.

Conclusions—In this population-based study, patients with IBD were about 9 times more likely to develop HS than the general population, with a female predisposition.

Keywords

Hidradenitis suppurativa; acne inversa; risk factor; epidemiology; inflammatory bowel disease; Crohn's disease; ulcerative colitis

INTRODUCTION

Hidradenitis suppurativa (HS) also known as acne inversa, is a chronic, relapsing inflammatory skin disease, characterized by recurrent painful, deep seated nodules, sinus tracts and abscesses.¹ This typically affects regions rich in apocrine glands, such as the anogenital region, inguinal and axillary areas, and may also involve the nape of the neck, inframammary region, buttocks and gluteal cleft. The estimated prevalence in the general population is approximately 0.1–1%, with a significant female predilection.^{2, 3} HS typically affects young adults in their third decade of life. Established risk factors for HS include cigarette smoking and obesity.¹

The pathogenesis of HS is unclear; recent studies have demonstrated dysregulation of the interleukin (IL)-12/23 pathway and tumor necrosis factor- α (TNF- α), suggesting an immune-mediated origin.^{4, 5} There appears to be a strong association between inflammatory bowel diseases (IBD) and HS, based on clinical and histologic similarities (development of sinus tracts, granulomatous inflammation and scarring); limited epidemiological evidence, primarily from referral centers, suggests a higher prevalence of HS in patients with IBD, ranging from 16–23%.^{6, 7} However, referral center studies are prone to selection and detection bias, and generally tend to over-estimate disease risk. Although estimates from population-based studies would be more representative of the risk and prognosis of HS in IBD, such studies have previously been unavailable.

Therefore, we sought to estimate the incidence of HS in IBD, and describe clinical characteristics and management of HS in a population-based cohort of patients with IBD. By comparing the incidence rate of HS in IBD with the incidence of HS in the general population in Olmsted County, Minnesota, we also sought to estimate whether IBD is an independent risk factor for HS.⁸

METHODS

This study was approved by the Mayo Clinic and Olmsted Medical Center Institutional Review Boards. As per Minnesota state law, we did not include patients who had withdrawn authorization to review their medical records for research purposes.

Setting

Olmsted County, situated in southeastern Minnesota, has a population of 144,260 inhabitants as of the 2010 US Census. Eighty-three percent of the population is non-Hispanic white, and a substantial proportion is of North European ancestry. The majority of people reside in Rochester, which is the urban center of an otherwise rural county. The residents of Olmsted County are socioeconomically comparable to the US white population, although a higher proportion are employed in health care services (25% in Olmsted County vs. 8% nationwide) and have a higher level of education (30% completed college in Olmsted County vs. 21% nationwide).^{9, 10}

Medical care within Olmsted County, Minnesota is practically self-contained within the region and is provided by Mayo Clinic, Olmsted Medical Center, and their affiliated hospitals, and the Rochester Family Medicine Clinic.¹¹ These healthcare providers are connected through a unique medical records-linkage system (the Rochester Epidemiology Project [REP]), which allows for the conduct of true population-based research studies. Each year, over 80% of the Olmsted County population is examined by one of the providers participating in the REP, and during any given 3-year period, greater than 90% of local residents are examined at one of the REP providers.¹¹ The linkage of information essentially enumerates the Olmsted County population; in fact, REP population estimates are consistently higher than those reported by the U.S. Census.¹² The central diagnostic index of REP comprises all diagnoses generated from outpatient evaluations, hospitalizations, emergency room evaluations, nursing home visits, surgical procedures, autopsy reports, and death certificates. It is therefore possible to identify all cases of a disease for which patients sought medical attention over a particular period of time.

Inflammatory Bowel Disease Evaluation

All potential new cases of CD and UC between January 1, 1970 and December 31, 2004 were identified through the central diagnostic index, and followed through death, emigration or the end of the study period (August 30, 2013). A diagnosis of CD was confirmed if at least two of the following criteria were satisfied, on two occasions, separated by at least 6 months: 1) clinical history of abdominal pain, diarrhea, weight loss, malaise, and/or rectal bleeding; 2) endoscopic findings of linear ulceration, mucosal cobblestoning, skip areas, or perianal disease; 3) radiologic findings of fistula, stricture, mucosal cobblestoning, or ulceration; 4) laparotomy appearance of “creeping fat,” bowel wall induration, and mesenteric lymphadenopathy; or 5) histologic findings of transmural inflammation and/or epithelioid granulomas. Likewise, a diagnosis of UC was made if a potential case satisfied the following criteria, on two occasions, separated by at least 6 months: 1) diffusely granular or friable colonic mucosa on endoscopy; and 2) continuous mucosal involvement as observed by endoscopy or barium studies. The phenotype of IBD was graded according to

the Montreal classification.¹³ We abstracted information on IBD therapy at time of diagnosis of HS and last follow-up, and classified therapy into sulfasalazine/5-aminosalicylate-based, corticosteroid-based, immunomodulator-based or anti-tumor necrosis factor α -based.

Hidradenitis Suppurativa Evaluation

Using the medical records linkage system, we identified all cases of HS in patients with IBD, diagnosed at outpatient clinic visits or hospitalizations, and pathology examinations, including autopsy. Subsequently, we manually reviewed all the medical records to verify the diagnosis, which was made based on a combination of clinical history and characteristic manifestations, in conjunction with an expert dermatologist, for cases with an unclear diagnosis.^{1, 14} We obtained information on the following disease characteristics: areas of HS involvement (axillae, inguinal area, thighs, perianal and perineal area, mammary and infra-mammary area, buttocks, pubic region and others); severity of lesions based on Hurley stage (stage 1 – abscess formation [single or multiple] without sinus tracts and cicatrization/scarring; stage 2 – recurrent abscesses with sinus tracts and scarring, single or multiple widely separated lesions; and stage 3 – diffuse or almost diffuse involvement, or multiple interconnected sinus tracts and abscesses across the entire area);¹⁵ treatment of HS (topical or oral antibiotics, topical or oral corticosteroids, hormonal therapy such as anti-androgenic therapy, biologic therapy with anti-TNF agents or ustekinumab, oral retinoids, other conventional immunosuppressive agents and/or surgery); associated dermatologic diseases and risk factors (smoking and obesity).

Statistical Analysis

Data were summarized as mean (standard deviation) or medians (interquartile range, IQR [25th percentile, 75th percentile]) for quantitative variables, and as frequency (%) for discrete variables. To estimate the incidence rate (IR) of HS in this population-based cohort of IBD patients, we recorded person-years at risk from the date of IBD diagnosis until the date of HS diagnosis, death, emigration or the end of the study period (August 30, 2013) for each individual in the cohort. To estimate the incidence rate ratio (IRR) of HS in IBD, we used a historical cohort of all HS patients identified in a general population-based study in Olmsted County.⁸ We calculated IRR by dividing the IR of HS in IBD with the IR of HS in the general population. The cumulative incidence of HS from the diagnosis of IBD was estimated using the Kaplan–Meier survival method. To assess the association of clinical characteristics in patients with HS and IBD (HS-IBD) (from our study) versus HS in the general population (HS-gen) (from the previous population-based study on HS in the general population from Olmsted County),⁸ we utilized two-sample t-tests (for continuous variables) or Fisher's exact test (for categorical variables).

All statistical analyses were conducted using SAS version 9.2 for Windows (SAS Institute Inc., Cary, NC). P-values <0.05 were considered statistically significant. Correction for multiple statistical comparisons was not made.

RESULTS

In our population-based inception cohort of 679 patients with IBD (313 with CD: age at diagnosis, 35.2 ± 18.2 y, 49.8% males; 366 with UC: age at diagnosis, 38.6 ± 17.1 y, 58.2% males), followed over a median of 19.8 (IQR, 0.2–43.4) years, we identified 8 patients with HS. Of these, one patient was diagnosed with HS prior to IBD diagnosis.

Incidence of Hidradenitis Suppurativa in IBD

The incidence of HS after IBD diagnosis was 0.55 cases per 1000 person-years, with comparable incidence in patients with CD (IR, 0.71) and UC (IR, 0.42). The 10-, 20- and 30-year cumulative probability of HS in IBD was 0.85% (95% confidence intervals [CI], 0.1–1.6%), 1.2% (95% CI, 0.2–2.2%) and 1.5% (95% CI, 0.3–3.5%), respectively. The overall prevalence of HS in IBD at the end of follow-up was 1.2%.

On comparing the incidence of HS in patients with IBD with the incidence reported in the general population from Olmsted County, we observed that the overall IRR was 8.9 (95% CI, 3.6–17.5). The IRR was comparable in patients with CD (IRR, 11.5) and UC (IRR, 6.8).

Clinical Characteristics of Hidradenitis Suppurativa in IBD

The clinical characteristics of patients diagnosed with HS are detailed in Table 1. The mean age of patients at time of HS diagnosis was 44.4 ± 8.3 years, and 7 out of 8 patients were females. Seven patients were smoking at time of diagnosis, and six were obese (median body mass index, 33.2 [IQR, 28.3–36.6] kg/m^2). None of the patients had a family history of HS. Axillae, groin and inner thighs were the most frequent sites of involvement. Three patients had a history of non-melanoma skin cancers (none arising in the area of HS involvement), and one patient had pyoderma gangrenosum. One patient, without a prior history of skin cancer, developed squamous cell cancer at the site of HS (in this patient the HS was diagnosed before the diagnosis of IBD).

Five patients with HS had underlying CD (including one patient diagnosed with HS before CD diagnosis). The median duration of CD before HS diagnosis was 8.4 years (IQR, 6.1–12.1). Two had ileocolonic disease and one patient had ileal disease. Four patients had associated perianal CD; two patients had penetrating phenotype, and the other two had non-penetrating, non-stricturing disease. Two patients were treated with sulfasalazine/5-ASA-based therapy, and one patient with a combination of corticosteroids, antibiotics and sulfasalazine; one patient was not on any IBD therapy.

Three patients with HS had underlying UC (median disease duration, 10 years [IQR, 9.6–15.3]) of whom two had undergone colectomy (with or without ileal pouch-anal anastomosis) by the time of HS diagnosis; one patient had ulcerative proctitis in clinical remission on 5-ASA. Of note, both the patients with colectomy developed pouchitis (one with antibiotic-responsive pouchitis, and another with antibiotic-refractory pouchitis requiring infliximab).

As compared with HS-gen in a historical cohort within Olmsted County, no significant differences in patient and clinical characteristics were observed in patients with HS-IBD

(Table 2). Patients with HS-IBD were similar to those with HS-gen, with regard to age, sex, BMI and smoking status. The primary sites of HS involvement were also comparable, with axillae, groin and thighs being the most common sites of involvement in both HS-IBD and HS-gen (axillae, 71.4% vs. 61.6%; groin or thigh, 57.1% vs. 69.4%); the perianal area was not more affected in patients with HS-IBD compared to HS-gen (14.3% vs. 10.1%; $p=0.53$).

Treatment of Hidradenitis Suppurativa

Two patients HS-IBD patients had Hurley stage 1 disease, limited to abscess formation, and were managed with oral antibiotics with or without incision and drainage; one patient required oral corticosteroids. Six patients had Hurley stage 2 disease, with recurrent abscesses, sinus tracts and scarring. These patients were managed using a variable combination of oral antibiotics and/or surgery; one patient required corticosteroids. None of the patients was treated with anti-TNF agents, systemic immunomodulators or anti-androgenic agents. As compared with HS-gen, HS-IBD patients had a trend towards more severe HS, although this observation was not statistically significant (Hurley stage II/III: HS-IBD vs. HS-gen, 71.2% vs. 40.3%; $p=0.13$).

DISCUSSION

In our population-based inception cohort study of 679 patients with IBD followed over a median of 19.8 years, we made several key observations. First, we observed that patients with IBD were about 9 times more likely to develop HS, as compared to the general population. This increased risk was comparable in both patients with CD (12 times higher risk, as compared to the general population) and UC (7 times higher risk). Second, we made novel observations on the IBD phenotype in patients with HS – most CD patients had perianal disease, and a majority of the UC patients who developed HS did so after colectomy, in the setting of pouchitis. Third, we did not observe significant differences in the HS phenotype in patients with IBD and the general population, although there was a trend towards more severe disease (higher Hurley stage). One patient with HS-IBD developed squamous cell skin cancer, an infrequent observation in the general population with HS.¹⁶ Finally, the diagnosis of HS usually followed the onset of IBD by nearly a decade in most patients (whereas HS preceded the development of IBD in only 1 of 8 patients). We also confirmed previous observations that female sex and obesity are established risk factors for HS.

Cross-sectional studies in the general population using chart reviews or self-reported questionnaires have suggested a prevalence of 0.1–1% in the general population.^{2, 3} In a cross-sectional survey of 599 participants from an unselected ‘healthy’ Danish population who offered a history and underwent physical examination, 1% were estimated to have clinical features compatible with HS.¹⁷ Similarly, in another population-based French survey of approximately 7000 participants, 67 self-reported HS (prevalence rate, 0.97%).² In contrast, clinic-based studies have overestimated the prevalence of HS – in a cross-sectional study of 507 patients undergoing screening at a sexually transmitted disease clinic, 4.1% were diagnosed with HS.¹⁷ In the only true population-based study on HS in the general population, from Olmsted County, Vazquez et al estimated the age- and sex-adjusted

incidence of HS at 6.0 cases per 100,000 person-years, with the highest incidence in females aged 20–29 years (IR, 18.4 per 100,000);⁸ the corresponding prevalence was 0.13%.³ There is a significant paucity of literature on the epidemiology of HS in IBD. In a cross-sectional interview of 158 IBD patients attending a meeting organized by the Dutch Crohn's and Ulcerative Colitis Patients Association (CCUVN), 16% reported a history suggestive of HS (17% amongst CD patients, 14% amongst UC patients).⁷ In another study amongst patients seen at an IBD clinic or members of CCUVN, using an non-validated self-reported questionnaire, van der Zee and colleagues reported a HS prevalence rate of 23% among IBD patients (26% amongst CD patients, 18% amongst UC patients).⁶ Ours is the first population-based study on the incidence and prevalence of HS in patients with IBD. We estimated that the incidence of HS in our population-based inception cohort of IBD patients at 0.55 cases per 1000 person-years, with the corresponding point prevalence of 1.2% (assuming HS to be a chronic disease). This incidence was comparable in patients with CD and UC. When compared to the incidence of HS in the general population as observed in a recent population-based study from Olmsted County,⁸ we estimated the relative risk of HS in IBD to be about 9 times higher, thus establishing IBD to be a strong risk factor for HS. While such inferences have been suggested based on indirect comparison of prevalence of HS in the general population and IBD across studies, this is the first time that this association has been objectively documented.

We observed that among 4 patients with CD and HS for whom phenotype information was relevant (excluding one patient who was diagnosed with HS before CD diagnosis), all had perianal disease and 2 had penetrating phenotype at time of CD diagnosis. Similarly, of the 3 patients diagnosed with UC and HS, 2 had undergone proctocolectomy with ileal pouch-anal anastomosis by the time of HS diagnosis for medically refractory disease, and both of these patients had active pouchitis. While based on small numbers, this may represent shared pathophysiology between HS and IBD. Such an association between IBD phenotype and HS has not previously been reported.

On comparing with a historical cohort of patients with HS-gen in Olmsted County, we observed that patients with HS-IBD may be older than patients with HS-gen (though not statistically significant), without significant differences in the sex distribution, and comparable distribution of other established risk factors (proportion with active smoking and obesity). As in the general population, female sex and obesity were risk factors for development of HS in patients with IBD.^{8, 17} HS location was also similar in patients with IBD and the general population; axillae, groin and/or thighs were the most common location. There was no specific predisposition towards perianal or perineal distribution in HS-IBD, as compared with HS-gen. In comparing across studies in different populations, van der Zee and coworkers observed that patients with HS-IBD were numerically (but not statistically) more likely to have HS in their buttocks (37% vs. 27%) and less likely in the perineal region (27% vs. 37%), as compared with HS-gen.⁶

Our study has several strengths: first, ours represents a well-characterized population-based inception cohort of IBD patients, in a defined geographic area with a stable population, over an extended period of time, and hence is truly representative of the general population; second, with well-characterized IBD phenotype, we were able to establish an association

between perianal CD and HS; third, the complete and long follow-up over 2 decades allows better assessment of rare outcomes such as HS. Our study also has several limitations: first, our sample size was fixed due to the population-based nature of this study, and hence, may have been underpowered for some detailed and comparative analyses; second, due to the retrospective nature of the disease, there was limited ability to assess the response of HS to therapy objectively; third, the population in Olmsted County is predominantly Caucasian (83%), which may limit the generalizability of the findings to other ethnicities. However, there is no suggestion that ethnicity predisposes to HS.

In conclusion, in our population-based inception cohort of 679 patients with IBD in Olmsted County, with a median follow-up of 19.8 years, we observed a nearly 9-fold increased risk of HS as compared to the general population. The risk was increased in both patients with CD and UC; interestingly, patients with HS were likely to have perianal CD, and in case of UC, have undergone colectomy with subsequent pouchitis. There also was a trend for patients with IBD to manifest more severe HS, as compared to the general population, although this was not statistically significant. Future prospective studies are warranted to understand the underlying shared pathophysiology of these conditions.

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Table 1

Characteristics of patients diagnosed with hidradenitis suppurativa

Patient ID	Age at HS diagnosis (in years); Sex	Body mass index (in kg/m ²)	Smoking status at time of HS diagnosis	IBD phenotype at time of HS diagnosis (Montreal); disease duration (in years)	IBD therapy at time of HS diagnosis	Location of HS involvement; Hurley stage	Treatment for HS
1	46.7; F	36.5	Yes	CD; A3, L3, B3+p; 7.3y	Sulfasalazine, prednisone, antibiotics	Axilla, groin/thigh; 2	Prednisone
2	47.7; M	21.0	Yes	CD; N/A (HS was diagnosed before CD)	N/A	Perineum, groin/thigh; 2	Oral antibiotics, surgery
3	45.6; F	22.2	Yes	CD; A3, L3, B3+p; 2.5y	Sulfasalazine	Axilla, groin/thigh; 2	Surgery
4	34.0; F	37.0	Yes	CD; unclear phenotype; 20.0y	None	Axilla; 2	Surgery
5	46.7; F	46.7	Yes	UC, s/p colectomy (antibiotic-responsive pouchitis); 9.3y	None	Axilla, groin/thigh, chest; 1	Oral antibiotics + Prednisone
6	45.5; F	32.4	Yes	UC, s/p colectomy (antibiotic-refractory pouchitis); 20.5y	Anti-TNF α (Infliximab)	Axilla; 2	Oral antibiotics
7	59.0; F	30.3	No	UC, E1, S0; 10.0y	5-aminosalicylate	Perineum; 1	Oral antibiotics
8	30.0; F	34.0	Yes	CD; A2, L1, B1+p; 9.4y	None	Groin/thigh; 2	Oral antibiotics, surgery

Note — IBD phenotype was defined based on Montreal classification at time of diagnosis of hidradenitis suppurativa [Abbreviations: CD-Crohn's disease; F-female; M-male; s/p-status post; TNF α -tumor necrosis factor; UC-ulcerative colitis; y-years]

Comparison of characteristics of HS in patients with IBD and in the general population (please note, that for this analysis, we only included patients who were diagnosed with HS after IBD diagnosis).

Table 2

Characteristics	HS-IBD (n=7)	HS-general population* (n=268)	p-value
Age	43.9±9.5	32.9±12.6	0.49
Sex (females/total)	7/7	189/268	0.20
Obesity (obese/total)	7/7	140/255	0.52
Smoking	6/7	153/265	0.24
Site of involvement			
Axillae	5/7	165/268	0.71
Groin/thigh	4/7	186/268	0.68
Perianal	1/7	27/268	0.53
Hurley stage II/III	5/7	108/268	0.13

* Derived from population-based study on incidence of hidradenitis in Olmsted County.⁸