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Conflicts of interest

None disclosed.

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Impact of psychiatric disease and treatment on biologic drug survival in patients with hidradenitis suppurativa

Biologic therapies have improved hidradenitis suppurativa (HS) treatment options[1]; yet, their limited drug survival remains a concern, with few studies exploring discontinuation reasons.[2] Psychiatric disease (PD) impairs the adherence to treatment for chronic diseases.[3] Hence, we assessed biologic drug survival and discontinuation risk in patients with HS with and without PD, stratified by active psychopharmacotherapy treatment.

We conducted a nationwide cohort study between 2005 and 2022, using the Danish national registries, including all patients with a HS diagnosis initiated on a biologic treatment. PD was defined as a hospital diagnosis of depression, anxiety, or schizophrenia, or ≥3 relevant psychopharmacotherapy prescriptions. Patients were considered under active treatment if they filled a psychopharmacotherapy prescription 12 months prior to biologic initiation. Discontinuation risk was assessed using age- and sex-adjusted hazard ratios (HR) from Cox regression models. Additional methodological details are provided in the Supplementary Material, available via Mendeley at https://data.mendeley.com/datasets/w4n8hybbpz/1.

Among 584 patients (1040 treatment series), 255 (43.7%) had a history of PD. Patients with PD were slightly older (mean age 42.0 [SD 12.3] vs 41.3 [SD 13.8]) and more often female (68.2% vs 53.3%) compared to those without PD (Supplementary Table I, available via Mendeley at https://data.mendeley.com/datasets/w4n8hybbpz/1). History of smoking (42.0% vs 28.4%), alcohol abuse (20.0% vs 13.5%), and recreational drug use (20.4% vs 13.5%) was more common in the PD group.

Median drug survival for patients with and without PD was 33.0 vs 36.9 weeks, respectively (Supplementary Fig 1, available via Mendeley at https://data.mendeley.com/datasets/w4n8hybbpz/1). Patients receiving psychopharmacotherapy had similar drug survival to patients with no PD (34.9 weeks, $P = .34$), and no increased discontinuation risk (HR = 1.01 [0.85-1.18]) (Table I). Patients not receiving psychopharmacotherapy had shorter drug survival (28.9 weeks, $P = .03$) and higher

Table I. Drug survival and risk of discontinuation of a biologic drug, both overall, and by depression, anxiety, schizophrenia and whether the patient had more than 1 psychiatric disease. Data shown for overall groups and stratified for whether the patients where currently receiving pharmacotherapy for their PD

Disease strata	Years of follow-up	Number of discontinuations	IR per PY (95% CI)	Adjusted HR*	95% CI	*P*-value
Overall	990.9	831	0.84 (0.78-0.90)			
No PD	563.6	445	0.79 (0.72-0.87)	[Reference]		
Any PD	427.3	386	0.90 (0.82-1.00)	1.08	0.94-1.24	.27
Depression	319.3	295	0.92 (0.82-1.04)	1.11	0.95-1.29	.17
Schizophrenia	98.0	76	0.77 (0.61-0.97)	0.99	0.77-1.26	.91
Anxiety	325.1	298	0.92 (0.82-1.03)	1.08	0.93-1.26	.28
> 1 PD	236.5	219	0.92 (0.81-1.06)	1.11	0.95-1.31	.20
Untreated PD						
Any PD	169.6	174	1.03 (0.88-1.19)	**1.22**	**1.02-1.45**	**.032**
Depression	107.7	117	1.09 (0.90-1.30)	**1.25**	**1.02-1.54**	**.031**
Schizophrenia	10.1	11	1.09 (0.54-1.95)	1.23	0.67-2.23	.51
Anxiety	119.0	124	1.04 (0.87-1.24)	**1.24**	**1.01-1.52**	**.036**
> 1 PD	62.0	73	1.18 (0.92-1.48)	**1.34**	**1.04-1.72**	**.022**
Current psychopharmacotherapy						
Any PD	257.7	212				
Depression	211.6	178	0.82 (0.72-0.94)	1.01	0.85-1.18	.98
Schizophrenia	87.9	65	0.84 (0.72-0.97)	1.03	0.87-1.24	.69
Anxiety	206.0	174	0.74 (0.57-0.94)	1.05	0.74-1.24	.74
> 1 PD	174.4	146	0.84 (0.72-0.98)	1.00	0.84-1.20	.93

Statistical significant differences shown in bold.
CI, Confidence interval; *HR*, hazard ratio; *IR*, incidence rate; *PD*, psychiatric disease, *PY*, person years.
*Adjusted for age and sex.

discontinuation risk (HR = 1.22 [1.02-1.45]) than those without PD (Supplementary Fig 2, available via Mendeley at https://data.mendeley.com/datasets/w4n8hybbpz/1). Patients with prior psychiatric hospitalizations showed significantly shorter drug survival (28.9 weeks, $P = .03$) and higher discontinuation risk (HR = 1.24 [1.02-1.50]) compared to patients without PD (Supplementary Tables II and III, available via Mendeley at https://data.mendeley.com/datasets/w4n8hybbpz/1). This effect was more pronounced in those hospitalized not receiving active psychopharmacotherapy, with a median survival of 24.9 weeks ($P = .03$) and an increased discontinuation risk (HR = 1.41 [1.03-1.93]) (Fig 1).

We found an overall PD history in 43.7% of patients, which is higher than prior estimates[4,5] likely due to more severe disease and higher comorbidity burden in patients starting biologics. Additionally, PD may impair adherence to prior HS treatments, potentially contributing to the initiation of biologics due to more severe disease trajectories.

Patients with PD on psychopharmacotherapy had drug survival similar to those without PD, while untreated PD was linked to significantly shorter drug survival. Psychiatric hospitalization predicted the poorest outcomes, likely reflecting more severe PD and warranting greater clinical attention for this group of patients.

A strength of this study is the large number of biologic treatment series as well as the long follow-up. Limitations include missing data on discontinuation reasons and potential underestimation of PD due to reliance on hospital diagnoses and prescriptions.

Overall, untreated PD decreases biologic drug survival in HS, an effect which is mitigated by psychopharmacotherapy. An integrated dermatologic-psychiatric approach may enhance biologic drug survival and improve patient outcomes.

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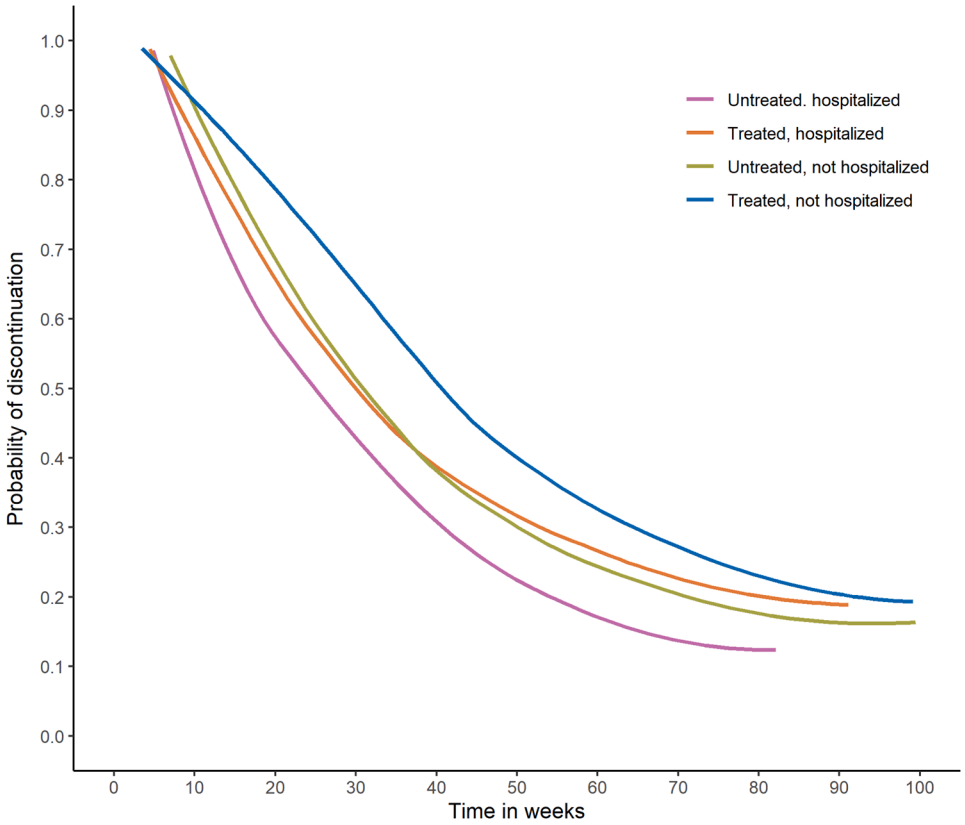

Fig 1. Kaplan-Meier curve (smoothened) for drug survival of biologic drugs in the intra group analysis of patients with HS and psychiatric disease (PD), for the first 2 years after initiation. Data stratified by history of hospital admission for PD and whether the patients are currently receiving psychopharmacotherapy. *Orange*: patients with a history of hospital admission for PD and currently receiving pharmacotherapy. *Blue*: patients not previously hospitalized for PD in current psychopharmacotherapy. *Green*: patients not previously hospitalized for PD not in current psychopharmacotherapy. *Pink*: patients previously hospitalized for PD not in current psychopharmacotherapy.

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Conflicts of interest

Nikolaj Holgersen and Valdemar Wendelboe Nielsen have no potential conflicts of interests regarding this paper. Hans-Christian Ring has received honoraria as a speaker/consultant for Abbvie, UCB, Novartis, Pierre Fabre and Janssen and is a principal investigator for Novartis, Janssen, Incyte, UCB and AbbVie. Prof. Ingram: received a stipend as immediate past-Editor-in-Chief of the British Journal of Dermatology and an authorship honorarium from UpToDate. He is a consultant for Abbvie, Boehringer Ingelheim, Cantargia, ChemoCentryx, Citryll, Elasmogen, Engitix, Incyte, Indero, Insmed, Kymera Therapeutics, MoonLake, Novartis, UCB Pharma, UNION Therapeutics, and Viela Bio. He is co-copyright holder of HiSQOL, Investigator Global Assessment and Patient Global Assessment instruments for HS and his department receives income from copyright of the Dermatology Life Quality Index (DLQI) and related instruments. Prof. Thyssen was previously an advisor for AbbVie, Almirall, Arena Pharmaceuticals, OM Pharma, Aslan Pharmaceuticals, Union Therapeutics, Eli Lilly & Co, LEO Pharma, Pfizer, Regeneron, and Sanofi-Genzyme,

a speaker for AbbVie, Almirall, Eli Lilly & Co, LEO Pharma, Pfizer, Regeneron, and Sanofi-Genzyme, and received research grants from Pfizer, Regeneron, and Sanofi-Genzyme. He is currently an employee of LEO Pharma. Prof. Egeberg has received research funding from Almirall, Pfizer, Eli Lilly, Novartis, Bristol-Myers Squibb, AbbVie, Janssen Pharmaceuticals, Boehringer Ingelheim, the Danish National Psoriasis Foundation, the Simon Spies Foundation, and the Kgl Hofbundtmager Aage Bang Foundation, and honoraria as consultant and/or speaker from Amgen, AbbVie, Almirall, Leo Pharma, Zuellig Pharma Ltd., Galápagos NV, Sun Pharmaceuticals, Samsung Bioepis Co., Ltd., Pfizer, Eli Lilly and Company, Novartis, Union Therapeutics, Galderma, Dermavant, UCB, Mylan, Bristol-Myers Squibb, McNeil Consumer Healthcare, Horizon Therapeutics, Boehringer Ingelheim, and Janssen Pharmaceuticals, and is currently employed by LEO Pharma. Simon Francis Thomsen has received research support from AbbVie, Almirall, Janssen, LEO Pharma, Novartis, Sanofi and UCB, and has been a speaker/consultant for Abbvie, Almirall, Boehringer, CSL, Dr August Wolff, Eli Lilly, Galderma, Incyte, Janssen, LEO Pharma, Novartis, Pfizer, Sanofi, Servier, Symphogen, UCB, and Union Therapeutics.

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Investigation of concordance between scalp symptoms, disease severity, and inflammatory activity in scarring alopecias

Alopecias can cause significant physical discomfort and psychosocial distress.[1] Scarring alopecias (SA) are characterized by permanent hair follicle damage, warranting prompt intervention.[2] Scalp symptoms and clinical signs of inflammation are used to determine disease activity and inform management. However, symptoms and inflammatory activity may not be concordant or accurately predict prognosis. Notably, patients lacking symptoms and signs of inflammation have been reported to experience continued alopecia progression.[3] We aim to evaluate scalp symptoms upon patients' initial presentation to NYU for SA, concordance with alopecia severity and inflammation, and persistence of symptoms/inflammation despite anti-inflammatory therapy.

We conducted an IRB-approved cross-sectional retrospective study of NYU patients aged 18+ years with a SA diagnosis seen between January 1, 2009 and November 1, 2024. Analyses were performed in SAS; t-tests and $\chi 2$/Fisher's exact tests were used for quantitative and categorical data, respectively.

589 individuals were included (82.2% female, mean age 54.8, 50.9% White, 30.7% unknown, 14.1% Black, 1.7% Asian, 0.85% Middle Eastern) (Table I). The most common diagnoses were lichen planopilaris (LPP) (45.0%), frontal fibrosing alopecia (FFA) (48.6%), and central centrifugal cicatricial alopecia (CCCA) (15.1%); some patients had overlapping diagnoses. 50.1% of patients reported scalp symptoms—32.4% itch, 9.0% pain, and 8.7% burning. 37.3%, 25.9%, and 40.5% reported symptoms at initial presentation for LPP, FFA, and CCCA, respectively. 41.9% had no inflammation, while 31.6%, 22.4%, and 4.1% had mild, moderate, or severe inflammation, respectively. For LPP, 20.8%, 49.1%, 22.6%, 6.8%, and 0.8%, had Grade 1, 2, 3, 4, and 5 alopecia, respectively. For FFA, 8.0%, 42.3%, 48.6%, and 1.1%, had Grade 1, 2, 3, and 4 alopecia, respectively. 54.9% were on anti-inflammatory medications, most commonly topical steroids (33.5%), intralesional steroids (31.8%), and doxycycline (14.8%), while 39.1% were on hair growth (anti-androgen/vasodilatory) medications, most commonly topical minoxidil (21.7%), oral finasteride (11.2%), and oral minoxidil (5.8%).

For the LPP/FFA cohort, symptomatic patients had significantly more inflammation on trichoscopic exam ($P < .0001$) and specifically, greater perifollicular scale and erythema ($P < .0001$) compared to asymptomatic patients (Table II). These results reflect concordance between scalp symptoms and inflammatory activity. Furthermore, symptoms were more common in patients on anti-inflammatory therapy (43.7% versus 26.3%, $P < .0001$), and these individuals had more severe inflammation ($P = .0365$). Several hypotheses may explain this finding: patients with more active disease may be more likely to receive anti-inflammatory therapy, treatment duration may have been too short to achieve maximum efficacy, or current doses may