Clindamycin versus clindamycin plus rifampicin in hidradenitis suppurativa treatment: Clinical and ultrasound observations

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Background: Antibiotics are recognized as first-line treatments for hidradenitis suppurativa (HS), but the data on their efficacy are limited.

Objective: Evaluate the efficacy of oral clindamycin versus that of clindamycin plus rifampicin in patients with HS.

Methods: A total of 60 patients with mild-to-moderate-severe HS who were classified according to their International Hidradenitis Suppurativa Severity Score System (IHS4) and Hurley scores, were subdivided into 2 groups of 30 patients each (group A, the members of which received clindamycin plus rifampicin, and group B, the members of which were treated with clindamycin alone) and retrospectively studied. The main objective was to evaluate and compare the clinical and ultrasound responses between the groups after 8 weeks of treatment according to the Hidradenitis Suppurativa Clinical Response measure.

Results: After the treatment, 17 of 30 patients in group A and 19 of 30 in group B met the primary outcome. Both groups showed a similar improvement of IHS4 score, whereas the Dermatology Life Quality Index and pain Visual Analogue Scale scores improved more in group B. In particular, the reductions in nodule and abscess counts were similar between the 2 groups, whereas the number of draining tunnels decreased more in group B. The factors significantly associated with Hidradenitis Suppurativa Clinical Response score were age, body mass index, IHS4 score, and absence of axillary involvement. Disease-free survival was similar between the 2 groups.

Limitations: The study was not randomized or placebo-controlled.

Conclusion: Clindamycin may be a useful treatment alternative to antibiotic combination regardless of HS clinical stage. (J Am Acad Dermatol 2019;80:1314-21.)

Key words: clindamycin; HS treatment; rifampicin.

Hidradenitis suppurativa (HS) is a chronic, inflammatory, recurrent, debilitating follicular skin disease that usually occurs after puberty. It is associated with painful deep-seated, inflamed lesions, most commonly in the axillae, inguinal, and anogenital region, and it has a great impact on the patient's quality of life.[1,2] Although the etiopathogenesis of the disease remains unclear, dysregulation of the pilosebaceous unit and an altered immune response have been recognized as crucial pathogenic factors for the onset of HS.[3] Though HS is not primarily an infectious disease,

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oral clindamycin and oral rifampicin are generally the first-line treatments for HS and are used for their antimicrobial, anti-inflammatory, and immunomodulatory properties.[4-9] However, antibiotic therapies may induce several adverse events, and a rapid emergence of resistant bacteria is occurring worldwide owing to the overuse and misuse of these drugs.[10-13]

The aim of the study was to assess and compare the clinical efficacy of clindamycin versus that of clindamycin plus rifampicin in patients with HS after 8 weeks of treatment according to the Hidradenitis Suppurativa Clinical Response (HiSCR) measure.

CAPSULE SUMMARY

- The aim of the study was to assess and compare the clinical efficacy of clindamycin versus that of clindamycin plus rifampicin in patients with hidradenitis suppurativa after 8 weeks of treatment according to the Hidradenitis Suppurativa Clinical Response measure.
- The study results suggest that clindamycin as monotherapy may be a useful and safe alternative to the combination of clindamycin and rifampicin regardless of the clinical stage of hidradenitis suppurativa.

MATERIAL AND METHODS

Patients

We performed a retrospective analysis of patients with HS who were seen in our department and treated with clindamycin or clindamycin plus rifampicin between June 2016 and June 2018. Patients were included in this retrospective analysis if they met all of the following criteria: male or female age 18 years or older; positive for anamnestic, clinical, and sonographic criteria of HS; total of 3 or more abscesses and/or inflammatory nodules at the baseline visit; and at least 1 year of follow-up. Furthermore, patients could not have either of the following exclusion criteria: prior treatment with clindamycin and/or rifampicin and other active skin disease or condition (eg, bacterial, fungal, or viral infection) that could interfere with assessment of HS. The following data were collected: sex, age, body mass index (BMI), localization of the lesions, disease duration as recalled by the patient, smoking history, comorbidities, Hurley score, International Hidradenitis Suppurativa Severity Score System (IHS4) score, Dermatology Life Quality Index (DLQI) score, Pain Visual Analogue Scale (VAS) score, Sonographic Scoring of Hidradenitis Suppurativa stage, and HiSCR measure.

PD ultrasound assessments

Power Doppler (PD) ultrasound examinations were performed on all patients at baseline and at week 8, with the limitation of intraobserver and interobserver reproducibility of PD ultrasound. To reduce PD ultrasound observation limitations and statistical mistakes, the pulse repetition frequency, time gain compensation, and volumetric image acquisition were kept constant. The analyzed sonographic data included detection of lesional type of involvement (nodule, abscess, or draining tunnel), lesion location, maximum diameter (in mm) of the lesion, thickness of the draining tunnel, and presence of vascularization. The longest draining tunnel was selected in patients with multiple and/or communicating draining tunnels. The lesions were categorized according to their sonographic morphology, and PD ultrasound criteria were assessed for diagnosing HS.[14-17] Patients were clinically and sonographically examined at all symptomatic sites, and the evaluation body sites considered were axillary, thoracic, inguinal, gluteal (including the intergluteal region), and occipitocervical. All examinations were performed by the same investigator using a MyLabOne ultrasound machine (Esaote, Genova, Italy) with a 10- to 18-MHz linear transducer.

Study design

The patients were divided into 2 groups according to the treatment received (Fig 1). Group A comprised patients treated with oral clindamycin, 150 mg 4 times a day, plus oral rifampicin, 300 mg 2 times a day, whereas group B consisted of patients treated with oral clindamycin, 150 mg 4 times a day. Both groups received antibiotic therapy for 8 weeks only. Patients were instructed to contact our department in the event of no clinical improvement. They attended a follow-up visit after completing the antibiotic therapy. Patients achieving HiSCR at the end of the treatment continued follow-up visits every 16 weeks. In contrast, patients from both groups who did not reach HiSCR at week 8 or had a disease relapse only after the end of therapy, were seen and retreated with oral clindamycin 150 mg 4 times a day, plus oral rifampicin, 300 mg 2 times a day, or adalimumab, or with other systemic therapies according to disease severity, their general condition, and the guidelines.

Assessment

The primary outcome was the clinical response after 8 weeks of antibiotic treatment, as defined

Abbreviations used:

BMI:	Body mass index
DLQI:	Dermatology Life Quality Index
HiSCR:	Hidradenitis Suppurativa Clinical Response
HS:	Hidradenitis suppurativa
IHS4:	International Hidradenitis Suppurativa Severity Score System
IL:	interleukin
PD:	Power Doppler
VAS:	Visual Analogue Scale

according to the HiSCR measure (as at least a 50% reduction from baseline in the total abscess and inflammatory nodule count, with no increase in the abscess or draining tunnel count). The following secondary outcomes of response were evaluated at week 8: (1) variation in count, size, pain, and PD ultrasound signals of nodules, abscesses and draining tunnels; (2) influence of patient characteristics (age, sex, familiarity, BMI, and smoking); (3) influence of disease characteristics (severity, affected areas, and disease duration); and (4) disease-free survival after antibiotic therapy. Additionally, DLQI was assessed at baseline and at week 8.

Statistical analysis

Efficacy outcomes were analyzed according to the group assignments. For the primary outcome, the number and percentage of patients who had a clinical response according to the HiSCR measure were calculated for each study group. For the secondary outcomes, demographic and clinical characteristic of the patients at baseline were correlated with clinical response. Number and characteristics of the lesions were compared at baseline and at week 8.

The differences between clinical and demographic groups were analyzed with the use of an independent *t* test, χ^2 test, and Fisher exact test, as appropriate. The incidence of clinical resolution was compared between treatment groups by using the χ^2 test. Statistical analysis of the factors, associated with a clinical response at week 8, was performed by bivariate and multivariate regression. Significance was assessed at a *P* value less than 5% (.05) for regressions, χ^2 test, independent *t* test, and Fisher exact test. Statistical analysis was performed by using SPSS Statistics software (version 23.0, IBM Corp, Armonk, NY).

The institutional review board approved the study and waived the need for informed consent from patients. All examinations were performed under the Helsinki principles of medical ethics.

RESULTS

Study participants

A total of 60 patients with HS were included in this study. Each group (A and B) was composed of 30 patients. The baseline characteristics (Table I) were generally similar across the study groups, except for age of onset, which was significantly higher in group A than in group B ($P = .035$). Group A had more patients in Hurley stage III than group B did owing to more diffuse involvement across the affected areas; however, the total number of lesions and IHS4 score were similar in both groups. Comorbidities were noted in 50% of patients in group A and 46.7% of patients in group B (Table I).

Clinical response

After 8 weeks of treatment, the responses to antibiotics were similar in both groups ($P = .598$): 17 of 30 patients in group A (56.7%) and 19 of 30 in group B (63.3%) met the primary outcome of a clinical response (according to the HiSCR measure) (Table II). Similarly, there was no significant difference between the 2 groups in terms of improvement in IHS4 score at the end of treatment ($P = .510$) (Table II). Pain VAS score ($P = .038$) and DLQI ($P = .037$) showed a statistically higher decrease in group B than in group A (Table II). However, response to antibiotic therapy was significantly higher in patients in Hurley stages I and II than in Hurley stage III ($P < .001$) and in patients with mild and moderate HS according to their IHS4 score than in those with severe HS according to their IHS4 score ($P = .02$), regardless of the group to which patients belonged (Table II). Furthermore, 1 year after the oral antibiotic treatment, the disease-free survival was similar between group A and group B ($P = .125$) (Table II).

In both groups a higher reduction in nodule and abscess count than in draining tunnel count was observed ($P < .001$). In group A, the numbers of nodules and abscesses decreased by 53.7% and 67.4%, respectively, whereas the number of draining tunnels diminished by just 14.8% (Table III). In group B, we observed a 46.7% decrease in the number of nodules, 68.1% decrease in the number of abscesses, and 38.8% decrease in the number of draining tunnels (Table III). The reductions in nodule count ($P = .517$) and abscess count ($P = .938$) were not statistically different between the 2 groups, whereas the decrease in number of draining tunnels number was statistically greater in group B than in group A ($P = .002$) (Table III). In addition, the average reduction in size of the abscesses and draining tunnels was higher in group B (Table III). The

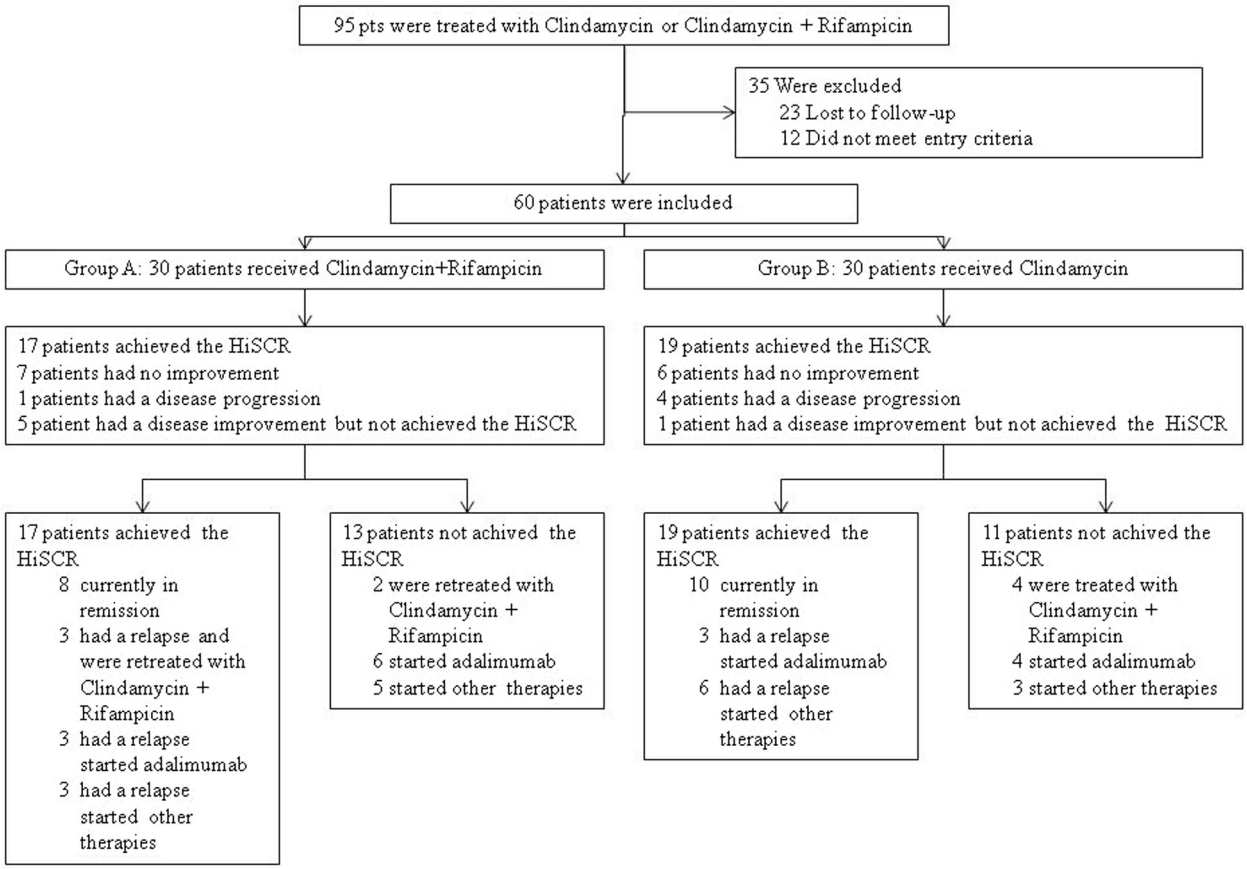

Fig 1. Study design. *HiSCR*, Hidradenitis Suppurativa Clinical Response.

numbers of nodules (P = .850) and abscesses (P = .904) with a positive PD ultrasound signal diminished similarly (without statistical differences) in the 2 groups, whereas the number of PD ultrasound–positive draining tunnels decreased more in group B than in group A, although the difference was not statistically significant (P = .085) (Table III).

The axillary areas and groin were the 2 most frequently affected sites in both groups (Table III). These 2 areas are also interesting in view of the high percentages of the total number of lesions—72.8% in group A and 71.7% in group B—that were located in these sites. The percentages of draining tunnels in these areas were even higher: 88.5% of those in patients in group A and 74.6% of those in patients in group B. A comparison of the affected area revealed the absence of a statistically different response in the 2 groups (P = .084 for group A and P = .089 for group B).

For the bivariate regression analysis, the factors significantly associated with the clinical response (according to the HiSCR measure) were age (coefficient, −0.46; P = .048), BMI (coefficient, −1.24; P = .011), and IHS4 score (coefficient, −0.107; P = .009) (Table IV). With the multivariate regression analysis, only axillary involvement (coefficient, −1.236; P = .048) and IHS4 score (coefficient, −0.80; P = .046) were significant (Table IV).

Safety

None of the 60 patients had any serious adverse events or discontinued the treatment. Diarrhea occurred in 6 of 30 patients in group A (20%) and 4 of 30 in group B (13.3%). In addition, 1 patient in group A reported candidal vaginitis.

DISCUSSION

Although HS is a chronic inflammatory skin disease, oral clindamycin and oral rifampicin are often the first-line treatments because of their antimicrobial, anti-inflammatory, and immunemodulatory properties.[4] Nevertheless, the information on their efficacy is very limited, with only 6 studies outlining the combination of oral clindamycin with oral rifampicin as an effective and tolerable regimen for most patients with HS existing to date.[4-9] However, antibiotic therapies may induce several adverse events.[10] In addition, rapid emergence of resistant bacteria is occurring worldwide as a result

Table I. Characteristic of the patients treated

Factors	Group A, n (%)	Group B, n (%)
Sex, n (%)		
Female	16 (53.3)	20 (66.7)
Male	14 (46.7)	10 (33.3)
Average age, y, ± SD	34.5 ± 12.7	31.7 ± 11.5
Age range, n (%)	18-57	18-63
Average BMI, kg/m^2, ± SD	28.6 ± 4.9	26.4 ± 7.1
Smokers, n (%)	21 (70)	24 (80)
Average age of onset, y, ± SD	22.5 ± 11.0	17.4 ± 7
Disease duration, y, ± SD	14.1 ± 8.9	15.3 ± 11.1
Family history, n (%)	6 (20)	4 (13.3)
Comorbidities, n (%)		
Acne	1 (3.3)	0
Psoriasis	1 (3.3)	1 (3.3)
Pilonidal cyst	1 (3.3)	1 (3.3)
Hashimoto's disease	6 (20)	3 (10)
PCOS	3 (10)	3 (10)
Thrombofilia (factor V Leiden or hyperhomocysteinemia)	3 (10)	1 (3.3)
Neurologic disorders (epilepsy, schizophrenia, and bipolar disorder)	1 (3.3)	2 (6.7)
Arterial hypertension	2 (6.7)	1 (3.3)
Diabetes	0	2 (6.7)
No comorbidities, n (%)	15 (50)	16 (53.3)
Hurley stage, n (%)		
I	5 (16.6)	6 (20)
II	11 (36.7)	19 (63.3)
III	14 (46.7)	5 (16.7)
Average IHS4 score ± SD	12.8 ± 10.8	13.6 ± 15.0
IHS4 category, n (%)		
Mild	2 (6.7)	1 (3.3)
Moderate	13 (43.3)	18 (60)
Severe	15 (50)	11 (36.7)
SOS-HS stage, n (%)		
I	0 (0)	3 (3.3)
II	14 (46.7)	12 (40)
III	16 (53.3)	15 (50)

BMI, Body mass index; *IHS4*, International Hidradenitis Suppurativa Severity Score System; *PCOS*, polycystic ovary syndrome; *SD*, standard deviation; *SOS-HS*, Sonographic Scoring of Hidradenitis Suppurativa.

Table II. Patients' disease score at baseline and at week 8 and HiSCR achievement

	Group A		Group B	
Score	T0	T8	T0	T8
Disease score at baseline and at wk 8				
Average IHS4 ± SD	12.8 ± 10.8	8.6 ± 11.9	13.6 ± 15.0	7.5 ± 12.7
Average Pain VAS ± SD	6.3 ± 2.3	4.2 ± 3.7	5.9 ± 2.5	2.5 ± 3.7
Average DLQI ± SD	15.2 ± 7.6	9.9 ± 9.4	14.3 ± 10.4	8.2 ± 10.8
Patients who achieved HiSCR				
Hurley stage I (%), n (%)	5/5 (100)		6/6 (100)	
Hurley stage II, n (%)	8/11 (72.7)		13/19 (68.4)	
Hurley stage III, n (%)	4/14 (28.6)		0/5 (0)	
IHS4 category mild, n (%)	2/2 (100)		1/1 (100)	
IHS4 category moderate, n (%)	9/13 (69.2)		15/18 (83.3)	
IHS4 category severe, n (%)	6/15 (40)		3/11 (27.3)	
No. of patients, n (%)	17/30 (56.7)		19/30 (63.3)	
Average disease-free survival, wk, ± SD	12.4 ± 12.2		13.2 ± 7.2	

DLQI, Dermatology Life Quality Index; *HiSCR*, Hidradenitis Suppurativa Clinical Response; *IHS4*, International Hidradenitis Suppurativa Severity Score System; *SD*, standard deviation; *VAS*, Visual Analogue Scale.

Table III. Characteristic of the lesion and areas affected at baseline and at week 8

Characteristic	Group A			Group B		
	T0, n (%)	T8, n (%)	Δ	T0, n (%)	T8, n (%)	Δ
Characteristic of the lesion at baseline and at week 8						
No. of nodules						
Total	41	19	22	45	24	21
PD	30	9	21	24	8	16
Average size, mm, ± SD	5.5 ± 3.1	5.7 ± 2.5	−0.2 ± 4	7.4 ± 4.8	7.6 ± 3.4	−0.2 ± 5.9
No. of abscesses						
Total	49	16	33	47	15	32
PD	46	16	30	41	15	26
Average size, mm, ± SD	14.0 ± 5.2	11.3 ± 8.7	2.7 ± 10.1	13.8 ± 4.6	7.7 ± 6	6.1 ± 7.6
No. of draining tunnels						
Total	61	52	9	67	41	26
PD	53	40	13	65	29	36
Average length, mm, ± SD	47,4 ± 21.7	36.5 ± 23.6	10.9 ± 32.1	43.8 ± 20.8	30.5 ± 23.2	13.3 ± 31.2
Average thickness, mm, ± SD	4.0 ± 2.1	3.8 ± 2.1	0.2 ± 3	4.1 ± 2.6	3.3 ± 2.4	0.8 ± 3.5
Total No. total of lesions	151	87	64	159	80	79

Areas affected at baseline at baseline and at week 8

Area	Type of lesion	Group A			Group B		
		T0, n (%)	T8, n (%)	Δ	T0, n (%)	T8, n (%)	Δ
Axillary	Nodules	6	5	1	11	6	5
	Abscesses	19	2	17	12	3	9
	Draining tunnels	31	29	2	27	22	5
	Total lesions	56	36	20	50	31	19
	No. of patients	21 (70)	16 (53.3)	5 (16.7)	11 (36.7)	10 (33.3)	1 (3.3)
Thoracic	Nodules	7	0	7	3	5	−2
	Abscesses	5	3	2	13	5	8
	Draining tunnels	2	2	0	7	3	4
	Total lesions	14	5	9	23	13	10
	No. of patients	6 (20)	4 (13.3)	2 (6.7)	6 (13.3)	5 (16.7)	1 (3.3)
Groin	Nodules	15	11	4	23	9	14
	Abscesses	16	7	9	18	6	12
	Draining tunnels	23	16	7	23	15	8
	Total lesions	54	34	20	64	30	34
	No. of patients	16 (53.3)	12 (40)	4 (13.3)	21 (70)	12 (40)	9 (30)
Gluteal	Nodules	12	3	9	8	4	4
	Abscesses	9	4	5	1	1	0
	Draining tunnels	5	5	0	9	0	9
	Total lesions	24	10	14	18	5	13
	No. of patients	9 (30)	6 (20)	3 (10)	5 (16.7)	1 (3.3)	4 (13.3)
Occipitocervical	Nodules	1	0	1	0	0	0
	Abscesses	0	0	0	3	1	2
	Draining tunnels	0	0	0	1	0	1
	Total lesions	1	0	1	4	1	3
	No. of patients	1 (3.3)	0 (0)	1 (3.3)	1 (3.3)	1 (3.3)	1 (3.3)

Δ indicates difference between T0 and T8.
PD, Power Doppler; *SD*, standard deviation.

of the overuse and misuse of these medications and a lack of new drug development. Among gram-positive pathogens (typical findings of HS lesions), a global pandemic of resistant species, including *Staphylococcus aureus* and *Enterococcus* spp, are now the biggest threats.[11,12] Resistance of *Streptococcus pneumonia* and *Mycobacterium tuberculosis* (including to isoniazid and rifampicin) is becoming epidemic.[12] Also, gram-negative pathogens are becoming resistant to nearly all available antibiotic drug options.

We studied the effectiveness of 8 weeks of oral antibiotic monotherapy versus that of combination antibiotic therapy to reduce the risk of side effects

Table IV. Logistic regressions

Factor	Coef	OR	95% CI	*P* value
Binomial regression				
Age	**−0.460**	**0.955**	**0.913-1**	**.048***
Disease duration	−0.340	0.967	0.918-1.018	.200
BMI	**−0.124**	**0.833**	**0.802-0.972**	**.011***
IHS4 score	**−0.107**	**0.899**	**0.830-0.973**	**.009***
Sex	−0.403	0.668	0.233-1.913	.452
Familiarity	0.525	1.690	0.391-7.308	.483
Smoking	0.365	1.441	0.443-4.690	.544
Multinomial regression				
Age	−0.340	0.967	0.906-1.032	.315
Disease duration	0.020	1.002	0.928-1.082	.965
BMI	−0.730	0.930	0.833-1.038	.195
IHS4 score	**−0.800**	**0.923**	**0.854-0.999**	**.046***
Sex	0.169	1.184	0.324-4.329	.798
Familiarity	−0.502	0.605	0.104-3.511	.605
Smoking	−0.493	0.611	0.147-2.532	.497
Axillary	**−1.236**	**0.290**	**0.085-0.991**	**.048***
Thoracic	−0.322	0.724	0.173-3.026	.659
Groin	−0.904	0.405	0.111-1.479	.405
Gluteal	0.132	1.141	0.296-4.392	.848
Occipitocervical	0.464	1.591	0.082-30.875	.759

Significant features are set in bold.
BMI, Body Mass Index; *CI*, Confidence interval; *Coef*, Coefficient; *IHS4*, International Hidradenitis Suppurativa Severity Score System; *OR*, Odds ratio.
*Significant ($P < .05$).

and promotion of resistant bacteria.[10,13,18,19] The results showed a statistical equivalence between monotherapy and combination therapy, with a significant improvement in disease activity in both groups, as assessed by the HiSCR measure, IHS4 score, PD ultrasound, Pain VAS score, and DLQI. Both protocols showed better results in relation to abscesses and nodules than in relation to draining tunnels, which is most likely related to draining tunnels' longer healing time and higher risk of relapse compared with those of nodules and abscesses. However, clindamycin alone seemed more effective in relation to draining tunnels than combination therapy was. Recently, 2 studies showed that rifampicin significantly decreases clindamycin concentration.[18,19] It could be speculated that the consequent lower clindamycin levels in combination protocols may reduce the strength of the treatment itself in cases of severe HS lesions, as draining tunnels are commonly colonized by polymorph-abundant anaerobic microflora.[20] Furthermore, a lower clindamycin level affects the long-term efficacy of the treatment and favors resistance.[20]

The sites involved did not appear to influence the degree of response, whereas the type of lesions (nodule, abscess, or draining tunnel) was correlated with the response rate. Pain VAS score and DLQI reduction were lower in group A than in group B, in which clindamycin gave better results in relation to draining tunnels, which are strongly related to pain and quality of life.

Young age, low BMI, low IHS4 score, and absence of axillary involvement were statistically correlated with HiSCR achievement. Younger patients attained HiSCR more frequently than older patients did. This association may be due to faster healing of the lesions, including draining tunnels, in young patients than in older patients. BMI has a strong connection with HiSCR; the results showed that patients with a low BMI had a better clinical response than did patients with a high BMI. In fact, obesity might influence HS pathogenesis in several ways owing to wider skin folds, enhanced friction, local sweat retention, or increased perspiration. In addition, obesity increases the levels of proinflammatory cytokines such as interleukin 1 (IL-1), IL-6, IL-8, tumor necrosis factor-α, and C-reactive protein, worsening the course of HS.[14] Additional data strongly related IHS4 score with HiSCR. It is known that HS is best treated by early intervention with medication aimed at reducing the development of draining tunnels, hypertrophic and/or atrophic-depressed scars with retraction and reduction of limb mobility, and therefore long-term disability. The data showed that patients with a low IHS4 score achieved HiSCR more easily than did patients with a high IHS4 score, who have an increased risk of treatment failure and disease relapse. In addition, axillary involvement seemed correlated with a reduced clinical response. Nonetheless, in our study draining tunnels were mostly localized in this area, and as stated earlier in this article, these lesions are the ones with the lower response to the treatment. These data might explain the correlation.

Finally, in our cohort, most patients treated with antibiotic therapy reached HiSCR, but 50% of patients had disease relapse after a mean time of 12.4 weeks and 13.2 weeks (from the end of the eight week treatment period) in group A and group B, respectively. This may indicate that antibiotic therapy does not cure the disease but just reduces the symptoms.

The mechanism of action of these 2 drugs in HS needs further research. Rifampicin is an antibiotic derived from *Streptomyces mediterranei*, which acts by binding to and inactivating bacterial DNA−dependent RNA polymerase but does not inhibit the mammalian enzyme.[21] It is a bactericidal agent and has broad-spectrum activity against the majority of gram-positive bacteria

(including *S aureus* and coagulase-negative staphylococci), as well as against many gram-negative microorganisms (including *Pseudomonas aeruginosa*) and *M tuberculosis* in particular.[22] However, bacterial resistance can occur rapidly when rifampicin is used as monotherapy.[13] Apart from its antimicrobial effects, it also modifies cell-mediated hypersensitivity by suppressing antigen-induced transformation of sensitized lymphocytes and T-cell function.[23] Clindamycin is a semisynthetic lincosamide antibiotic successor to lincomycin. It inhibits bacterial protein synthesis by binding to bacterial 50S ribosomal subunits. Clindamycin may be bacteriostatic or bactericidal depending on the organism and drug concentration. It is active against most anaerobic bacteria and gram-positive cocci except enterococci.[24] Also, clindamycin has the potential to modify or suppress inflammation. It suppresses the complement-derived chemotaxis of polymorphonuclear leukocytes in vitro, reducing inflammation.[25]

CONCLUSION

In conclusion, these results suggest that antibiotic therapy is an effective treatment of patients with HS and that clindamycin as monotherapy may be a useful and safe alternative to combination of the antibiotics clindamycin and rifampicin regardless of the clinical stage of HS. Prospective randomized controlled trials are needed to confirm these results.

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