



Original Article

5 Real-World Experience With Secukinumab for Hidradenitis Suppurativa: A
6 Multicenter Retrospective Analysis of 263 Patients From the SECU-SPAIN
7 Study

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ABSTRACT

Background/objective: There is no consensus on the response rates of secukinumab, as findings from phase III trials differ from those reported in clinical-practice studies. This study aimed to assess the mid-term safety and efficacy profile of secukinumab in patients with moderate-to-severe hidradenitis suppurativa (HS).

Methods: This retrospective, multicenter study included patients with moderate-to-severe HS treated with secukinumab (300 mg every four weeks) between 2020 and 2024. Eligible patients were aged ≥ 18 years, with a clinical diagnosis of moderate-to-severe HS, and a minimum follow-up of 24 weeks. Key exclusions included patients treated with nonstandard secukinumab dosing regimens or those with insufficient clinical data. The primary endpoints were achievement of HiSCR50 ($\geq 50\%$ reduction in the combined count of nodules and abscesses) at weeks 16 and 24. Secondary endpoints included achieving a $\geq 55\%$ reduction in the International HS Severity Score (IHS4-55) and adverse events.

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Results: A total of 263 patients (49.4%, women; 50.6%, men; mean age, 41.8 ± 13.2 years) were included. The most common HS phenotype was mixed (49.4%), and 55.1% had Hurley stage III disease. At weeks 16 and 24, a total of 57.4% and 63.6% of patients achieved HiSCR50, respectively. The mean IHS4 score dropped significantly from 16.7 ± 10.0 at baseline to 7.6 ± 5.6 at week 24 ($p < 0.0001$). At weeks 16 and 24, 52% and 56% of patients achieved an IHS4-55 response. Secukinumab was discontinued in 14.5% of patients because of lack of efficacy or adverse events.

Conclusions: Secukinumab demonstrated a significant safety and efficacy profile for patients with moderate-to-severe in a real-world setting, with better outcomes than those reported in earlier clinical trials. Response evaluation at both week 16 and 24 is crucial due to variations in treatment effectiveness.

38 Introduction

Q2 HS (HS) is a persistent, relapsing, inflammatory dermatological condition originating from pilosebaceous units, predominantly impacting intertriginous regions, and frequently correlated with multiple systemic comorbidities.¹ Due to its chronicity and recurrent episodes, HS significantly impacts the patients' quality of life, profoundly affecting social, occupational, and psychological dimensions.² In Europe, the reported prevalence of HS fluctuates from 1% to 4% across various studies, attributable to differences in study populations and methodologies³, while epidemiological data from American surveys report prevalences between 0.05% and 0.20%.^{4,5}

HS is clinically marked by recurrent episodes of neutrophilic inflammation. The inflammation initiates in the hair follicles, leading to the formation of painful nodules and abscesses, which, in advanced stages, develop into pus-discharging tunnels and extensive scarring.^{1,3}

Although the pathogenesis of HS has not been fully understood, it is a multifactorial disease resulting from a combination of genetic, environmental, and immunologic factors.^{1,3,6-8}

The initial pathophysiological events in HS are thought to be driven by the release of pathogen-associated molecular patterns (PAMPs) and danger-associated molecular patterns (DAMPs) following early follicular occlusion and bacterial overgrowth. These events activate the inflammasome, leading to the secretion of Interleukin (IL)-1 β , primarily by tissue macrophages, and subsequent release of downstream cytokines, including IL-17 and TNF α (6-8). Altered Toll-like receptor (TLR) signaling in macrophages and dendritic cells (DCs), which are the predominant cells in HS lesions, results in the elevated production of these cytokines, which triggers the activation of DCs, which in turn secrete IL-23, promoting the polarization of Th17 cells. IL-17-producing T helper cells have been observed to infiltrate the dermis in chronic HS lesions.⁶⁻⁸

HS treatment depends on disease severity and individual impact, involving topical, systemic, surgical, and combined approaches.⁹ Topical therapy is preferred in early stages, while systemic therapies, such as antibiotics, anti-inflammatory agents, immunosuppressants, botulinum toxin, isotretinoin, and antiandrogens are used for more severe cases.^{6,10}

The introduction of biologics has transformed the management of moderate-to-severe HS. For years, adalimumab (a TNF- α inhibitor) was the only approved biologic.^{11,12} Recently, anti-IL-17 biologics, such as secukinumab, ixekizumab, brodalumab have emerged as options for patients unresponsive to other therapies.¹³⁻¹⁵

Data from 2 double-blind, randomized phase III clinical trials (SUNSHINE and SUNRISE) have demonstrated the safety and efficacy profile of secukinumab for treating moderate-to-severe HS vs placebo, with 42–48% of patients achieving HS Clinical Response (HiSCR).¹⁶⁻¹⁸ Additionally, different clinical-practice studies have shown that secukinumab may be effective in treating HS, although the proportion of treatment-responders was variable.¹⁹⁻²⁷

The current study aimed to assess the mid-term safety and efficacy profile of secukinumab in patients with moderate-to-severe HS.

89 Methods

Study design

We conducted a retrospective and multicenter study under real-world conditions, including consecutive HS patients with moderate-to-severe HS²⁸ who underwent treatment with secukinumab from 2020 through 2024. The study ensured a minimum follow-up period of 24 weeks, with a maintenance dose of 300 mg administered every 4 weeks.

The study protocol was approved from IIS La Fe Ethics Committee (Valencia, Spain), which waived the requirement for informed consent to conduct the study. However, all participants gave their prior written informed consent before receiving secukinumab. This study fully complied with the Good Clinical Practice/International Council for Harmonization Guidelines, the Declaration of Helsinki, and all applicable country-specific regulations governing clinical research, prioritizing whichever provided greater protection to the individual. All identifying information was encrypted or withdrawn to ensure participant anonymity.

Study population

Patients aged ≥ 18 years old with a clinical diagnosis of moderate-to-severe HS, who underwent treatment with secukinumab, with a maintenance dose of 300 mg administered every 4 weeks, and had a minimum 24-week follow-up. We excluded those patients with other secukinumab posology and cases with a lack of the data needed to analyze the therapeutic response.

Patients were required to have a confirmed diagnosis of HS for at least 6 months prior to study initiation, in accordance with the inclusion criteria defined in the SUNNY trials.¹⁶⁻¹⁸

Outcomes

The primary endpoints were HiSCR50 (defined as the proportion of patients who achieved at least a 50% reduction in nodule and abscess count) at week 16 and week 24.

Secondary endpoints were the proportion of patients who achieved a $\geq 55\%$ reduction of the International HS Severity Scoring System (IHS4-55) score²⁹ and the safety profile (in terms of treatment-related adverse events).

Clinical phenotypes of HS were defined according to Martorell et al. classification.³⁰

Pain was determined according to the Numerical Rating Scale (NRS)⁹; however, due to the limited number of patients with available data ($n = 12$), this variable was excluded from the statistical analysis.

Statistical analysis

A standard statistical analysis was performed using SPSS Statistical Software version 28 (IBM SPSS Statistics, IL, United States).

Table 1**Q4** Key demographic and clinical features of the study cohort.

Variable	
Age, years	
Mean \pm SD	41.8 \pm 13.2
Sex, n (%)	
Women	130 (49.4)
Men	133 (50.6)
BMI (kg/m²)	
Mean \pm SD	23.4 \pm 4.6
Family history of HS, n (%)	
Yes	78 (29.7)
No	185 (70.3)
Course of the disease, years	
Mean \pm SD	15.4 \pm 9.2
Hurley stage, n (%)	
I	8 (3.0)
II	110 (41.8)
III	145 (55.1)
Phenotype, n (%)	
Mixed	130 (49.4)
Inflammatory	126 (47.9)
Follicular	7 (2.7)
Areas involved	
Mean \pm SD	5.1 \pm 2.2
Number of previous systemic treatments	
<i>Non-biologics</i>	
Mean \pm SD	4.1 \pm 2.1
<i>Biologics</i>	
Mean \pm SD	1.8 \pm 0.9
Type of previous systemic treatments, n (%)	
<i>Non-biologics</i>	
Systemic antibiotics ^a	263 (100.0)
Systemic corticosteroids ^b	84 (31.9)
Antiandrogens ^c	50 (19.0)
Oral antidiabetics ^d	31 (11.8)
Retinoids	
Isotretinoin	31 (11.8)
Acitretin	21 (8.0)
<i>Biologics</i>	
Adalimumab	244 (93.1)
Infliximab	31 (11.8)
Ustekinumab	21 (8.0)
Guselkumab	16 (6.1)
Risankizumab	13 (4.9)
Number of previous surgical procedures*	
Mean \pm SD	2.5 \pm 2.8
Inflammatory nodules	
Mean \pm SD	3.5 \pm 4.1
Abscesses	
Mean \pm SD	1.8 \pm 2.0
Inflamed or draining tunnels	
Mean \pm SD	4.6 \pm 4.1
IHS4	
Mean \pm SD	16.7 \pm 10.0

Table 1*(Continued)*

Variable	
Pain NRS	
Mean \pm SD	5.4 \pm 3.5
Combine treatment, n (%)	
None	79 (30.1)
Antiandrogen therapy	29 (11.0)
Antidiabetic therapy	35 (13.3)
Systemic antibiotic	85 (32.3)
Systemic corticosteroids	39 (14.9)
Combined surgery, n (%)	
No	258 (98.1)
Yes	5 (1.9)
Dosage (maintenance dose), n (%)	
300 mg every 4 weeks	244 (92.8)

SD: standard deviation; BMI: body mass index; HS: HS; IHS4: International HS Severity Scoring System; NRS: Numerical Rating Scale.

^a Including combination therapy with rifampin 300 mg every 12 h and clindamycin 300 mg every 12 h for 10 weeks, or doxycycline 100 mg every 12 h for 12 weeks.

^b Including tapering prednisone regimen at 0.5–1 mg/kg for 15–30 days.

^c Spironolactone 50–100 mg/day.

^d Metformin.

* Excluding incision and drainage.

Descriptive statistics, including number (percentage) and mean \pm standard deviation (SD), were applied as appropriate.

Results

Baseline demographic and clinical characteristics

A total of 263 patients, 130 (49.4%) women and 133 (50.6%) men, were included in the study. Mean age was 41.8 \pm 13.2 years.

The most prevalent HS phenotype was the mixed type, observed in 130 (49.4%) patients, followed by the inflammatory phenotype in 47.9% of patients, and the follicular phenotype in 2.7%.

Regarding HS severity, a total of 145 (55.1%) patients exhibited Hurley stage III disease; 110 (41.8%) patients, Hurley stage II disease; and 8 (3.0%) patients, Hurley stage I disease.

The mean IHS4 score was 16.7 \pm 10.0. The mean number of previous systemic treatment were 4.1 \pm 2.1 and 1.8 \pm 0.9 drugs for the non-biologic and biologic therapies, respectively. Of the 263 patients included in the study, 18 (6.8%) were biologic-naïve at the time of treatment initiation. The mean number of previous surgical procedures, excluding incision and drainage was 2.5 \pm 2.8 procedures.

The main demographic and clinical characteristics are shown in Table 1.

Efficacy

HS clinical response (HiSCR)

The proportion of patients who achieved a HiSCR50 at week-16 and at week-24 of secukinumab treatment were 57.4% and 63.6%, respectively (Fig. 1A).

International HS Severity Score System (IHS4-55)

The mean IHS4 score significantly dropped from 16.7 \pm 10.0 points at baseline to 10.1 \pm 5.2 points at week 16 and 7.6 \pm 5.6 points at week 24, with both reductions being statistically significant ($p < 0.0001$ vs baseline).

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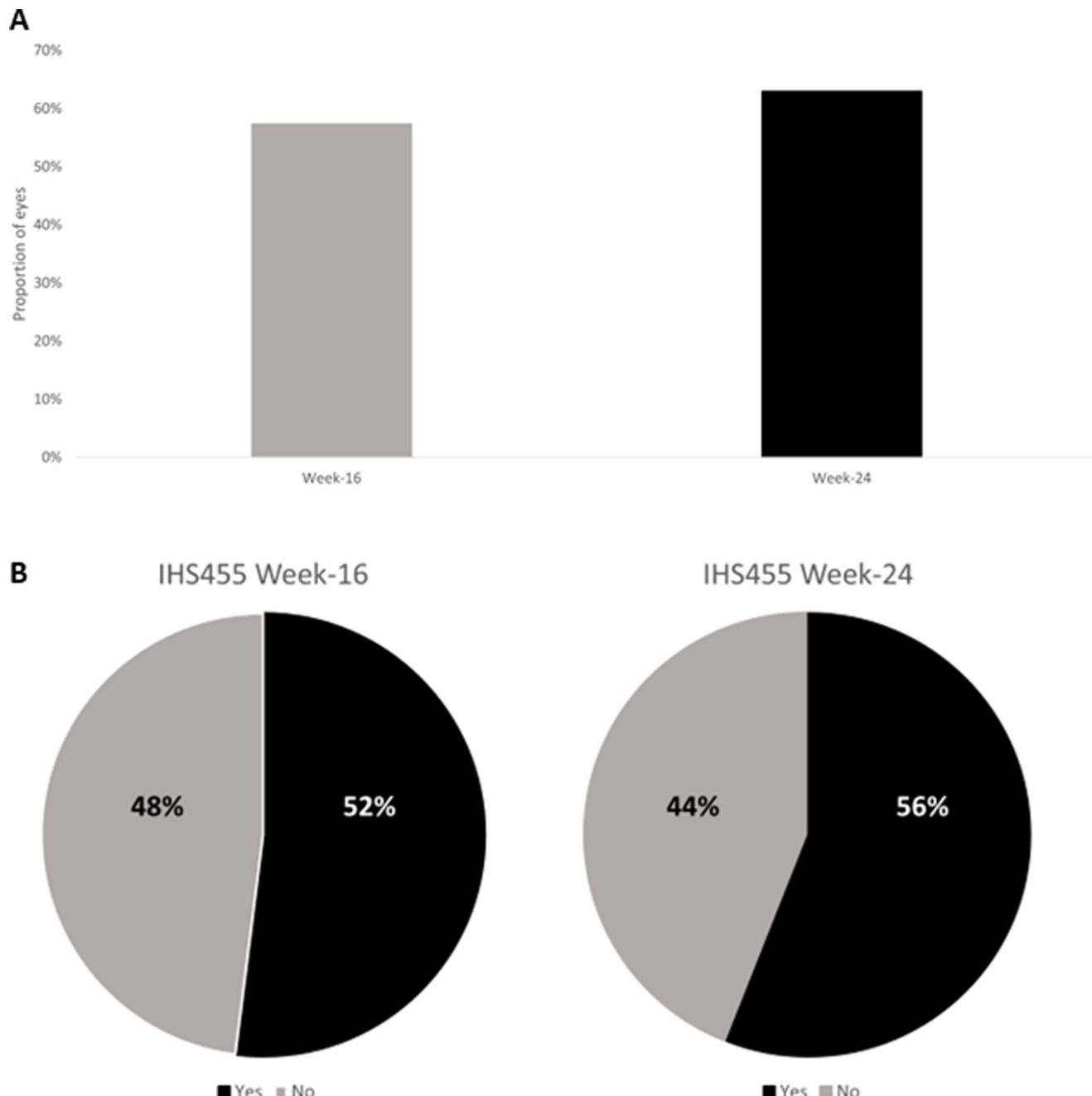


Fig. 1. Overview of the main study outcomes. (A) Proportion of patients who achieved a HiSCR50 16 and 24 weeks into secukinumab therapy. (B) Proportion of patients who achieved a 55% reduction of the International HS Severity Scoring System (IHS4) score at weeks 16 and 24, respectively.

162 At week 16 and 24 of secukinumab treatment, 52% and 56% of
 163 patients reached an IHS455 (Fig. 1B).

164 Compared with the administration of secukinumab alone, the
 165 combination therapy showed better results, regardless of the scale or time of
 166 measurement. At week 16, the proportion of patients achieving HiSCR50
 167 was significantly higher in the combination therapy group (62.2%) vs
 168 those on secukinumab monotherapy (52.2%) ($p < 0.05$). This
 169 difference persisted and slightly increased by week 24, with response rates
 170 of 66.7% vs 55.5%, respectively ($p < 0.05$).

171 A time-restricted subanalysis revealed statistically significant
 172 differences in treatment response based on baseline disease severity and
 173 duration. Patients with moderate disease (Hurley II) showed higher
 174 response rates vs those with severe disease (Hurley III), achieving
 175 HiSCR50 in 62.2% and 65.7% of cases at weeks 16 and 24, respectively,

176 and IHS4-55 in 68.1% at week 24. In contrast, Hurley III patients had
 177 lower rates: 48.3% (HiSCR50, week 16), 51.1% (week 24), and 52.3%
 178 (IHS4-55) ($p < 0.05$).

179 Similarly, shorter courses of the disease (<5 years) were associated
 180 with better outcomes: HiSCR50 responses were 63.3% at week 16 and
 181 68.4% at week 24, with 71.2% reaching IHS4-55. Patients with longer
 182 disease duration (>5 years) had lower response rates (51.2%, 53.4%,
 183 and 55.5%, respectively; $p < 0.05$). These findings underscore the influence
 184 of disease stage and chronicity on treatment efficacy.

Safety and treatment withdrawal

185 Throughout follow-up, 38 (14.5%) patients withdrew secukinumab
 186 due to lack of response ($n = 33$; 12.6%); oral candidiasis ($n = 1$, 0.4%)
 187

Table 2

A comparison of the clinical outcomes between the current study and the available real-world evidence.

Source	Sample size	HiSCR50	Baseline IHS4	IHS4 response	IHS4-55
Casseres et al. ²⁰	20	70% (24w)	N.A.	N.A.	N.A.
Melgosa et al., 2022 ²³	23	73.9% (16w) 83.3% (52w)	11.4 ± 9.3	5.8 ± 7.1 (16w) 4.73 ± 7.9 (52w)	N.A.
Fernández-Crehuet et al., 2023 ²⁴	47	48.9% (16w)	21.11 ± 11.9	12.7 ± 11.6 (16w)	40.4% (16w)
Martora et al., 2024 ²⁵	21	57.1% (16w) 71.4% (52w)	10.4	N.A.	N.A.
Haselgruber et al., 2024 ²⁶	67	41.8% (24w)*	N.A.	N.A.	44.8% (24w)
Current study	263	57.3% (16w) 63.6% (24w)	16.65 ± 10.02	N.A.	56.0% (24w)

N.A.: not available; w: week; m: minute; HiSCR: hidradenitis suppurativa clinical response; IHS4-55: proportion of patients who achieved a 55% reduction of the International HS Severity Scoring System (IHS4) score.

* Not calculable (<3 AN count) in 17.91% of patients.

188 psoriasis worsening ($n = 2$; 0.8%); ulcerous colitis flare ($n = 1$; 0.4%);
189 and Crohn's disease flare ($n = 1$; 0.4%).

190 Discussion

191 According to the results of the current real-world study, we con-
192 cluded that in clinical practice secukinumab demonstrates to be a safe
193 and effective treatment for patients with HS refractory to conventional
194 systemic therapy. Key highlights include the high proportion of patients
195 who achieved an IHS4-55 and HiSCR at week-16 and week-24 of treat-
196 ment, as well as the low rate of treatment discontinuation (12.6%).

197 The Sunny trials (Sunrise and Sunshine)¹⁶⁻¹⁸ were 2 randomized
198 control trials that assessed the safety and efficacy profile of secukinumab
199 in patients with moderated-to-severe HS over a period of 52 weeks. The
200 primary endpoint was the proportion of patients achieving HiSCR50,
201 which ranged from 42% (Sunrise) to 46% (Sunshine) in the treatment
202 groups.

203 Although HiSCR is a tool widely used in clinical practice for assess-
204 ing the reduction of inflammatory nodules and abscesses, it does not
205 include the reduction of draining tunnels in its evaluation.¹¹ Recently,
206 the IHS4-55 tool has been introduced for evaluating the efficacy of HS
207 treatments.^{28,31}

208 At week 16, 57.4% of patients achieved HiSCR50. In the same line,
209 at week 16 up to 52% of patients reached an IHS455 (Fig. 1B).

210 The results of our study suggested slightly improved outcomes vs
211 those reported by the "Sunny trials." Specifically, the proportion of
212 patients achieving HiSCR50 was 57.4% at week 16 (vs 42% in the
213 Sunrise and 46% in the Sunshine) and 63.6% at week 24 (vs 56% in the
214 Sunny).^{16,17}

215 The slightly better outcomes observed in our study may be attributed
216 to the fact that 69.9% of the sample received combination therapy.
217 Clinical trials on HS typically exclude treatments aimed at managing
218 comorbidities that could potentially influence patient outcomes.¹⁶⁻¹⁸ In
219 contrast, daily clinical practice and current clinical guidelines under-
220 score the importance of addressing comorbidities, as their effective
221 management positively impacts the control of HS.³²⁻³⁴

222 Although randomized clinical trials (RCTs) represent the highest
223 level of clinical evidence,³⁵ they have limitations that may reduce their
224 clinical validity and applicability to certain population groups. Clinical-
225 practice data can offer valuable insights into treatment efficacy across
226 diverse patient subgroups in clinical practice.^{36,37}

227 Other studies, conducted on daily clinical-practice conditions, have
228 assessed the effectiveness of secukinumab in HS, predominantly using
229 HiSCR as the primary outcome measure, with HiSCR rates ranging from
230 46% to 83.6% at 16-52-week follow-ups.¹⁹⁻²⁷ However, these studies
231 often involved limited samples (Table 2).

232 Casseres et al.²¹ found that 65% (13/20) of patients achieved HiSCR
233 at week 12. Similarly, Melgosa et al.²⁴ followed 23 patients and reported

234 that 73.9% (17/23) achieved HiSCR at week 16, with sustained results
235 of 71.4% (15/21) at week 24, 71.4% (10/14) at week 36, and 83.3%
236 (10/12) at week 52 of secukinumab treatment. Fernandez-Crehuet
237 et al.²⁵ reported that 48.9% of patients achieved HiSCR at week 16,
238 while Martora et al.²⁶ found that 57.1% (8/14) of patients achieved
239 HiSCR at week 16, with 71.4% (10/14) reaching HiSCR by week 52.

240 There is ongoing debate regarding the optimal time point for assess-
241 ing therapeutic response to biologic agents, with week 24 generally
242 considered the most informative in clinical practice. In our study, we
243 observed an increased response at this time point, with 63.6% of patients
244 achieving HiSCR50 and up to 56% achieving IHS4-55. These findings
245 contrast with those reported by Haselgruber et al.,²⁷ in a retrospective
246 study of 67 patients, where 41.79% (28/67) achieved HiSCR and
247 44.78% (30/67) achieved IHS4-55 at week 24.

248 To the best of our knowledge, the current study, which includes 263
249 patients, is the largest clinical-practice analysis of secukinumab for HS
250 conducted thus far.

251 Compared with published evidence, both clinical trials and clinical-
252 practice studies, the HiSCR rate in our study is in the upper range of
253 these studies. Nevertheless, it must be considered that the sample of
254 this study presented a high proportion of patients at the Hurley III stage
255 (indeed 97% of patients were either stage II or III), a long disease dura-
256 tion (15.4 ± 9.2 years), and that patients presented previous exposure to
257 biologic drugs (mean, 1.8 ± 0.9 drugs).

258 This study has limitations that should be considered when evaluating
259 its results. First, its retrospective design introduces potential confounding
260 variables and inherent biases. Nevertheless, a key strength of this study
261 is its execution within clinical settings, allowing for the inclusion and
262 analysis of patient scenarios that extend beyond those typically assessed
263 in controlled clinical trials. Secondly, its limited follow-up duration. A
264 longer follow-up period might provide more comprehensive insights.
265 Important strengths of this study include its multicenter design and the
266 high number of patients included in it.

267 Conclusions

268 The results of this multicenter real-world study indicate that secuk-
269 inumab can be considered a safe and effective treatment option for
270 patients with moderate-to-severe HS who have failed other therapies,
271 even with higher results vs the previous data published in the literature
272 from the Sunny clinical trials. Special consideration would be made in
273 the best moment to evaluate the response to medical therapy due to the
274 differences between week 16 and 24.

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278 Conflicts of interest

279 Antonio Martorell declared to have received honoraria and/or travel
 280 grants and/or acted as an advisory board member for Novartis, Abb-
 281 Vie, Janssen Cilag, UCB, Lilly, LEO Pharma, L'Oréal, Sanofi, Boehringer
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 288 for Novartis, Abbvie, Janssen Cilag, UCB, Lilly, LEO Pharma, L'Oréal,
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