Original Article

Evaluation of Super Response in Plaque-Type Psoriasis Patients Treated with IL-17 and IL-23 Inhibitors

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Abstract

Aim: Biologic treatments have become important for optimal disease control in patients with moderate to severe plaque psoriasis. The term "super response" is used to describe a faster and more sustained response to biologic treatment. This study aimed to determine the characteristics of non-super-responder (NSR) and super-responder (SR) patients with psoriasis receiving interleukin (IL)-17 or IL-23 inhibitors.

Materials and Methods: Patients with plaque psoriasis who were treated with IL-17 or IL-23 inhibitors between 2020 and 2024 were retrospectively analyzed. A super response was defined as a psoriasis area and severity index (PASI) 100 response at week 16 that was maintained through week 28. The patients were divided into the NSR and SR groups and compared in terms of sociodemographic characteristics, clinical findings, biological treatments, and lipid profiles.

Results: A total of 200 patients were included in the study; 96 (48%) were SRs. The frequency of super response was significantly higher among patients with an initial PASI score of ≥ 10 (P = 0.041). Compared with the SR group, the NSR group was more likely to have a history of biologic therapy (P = 0.002). Continued use of the same biological agent was more frequent among SRs (P = 0.019). In the multivariate logistic regression analysis, prior biologic therapy was independently associated with a lower likelihood of achieving a super response [odds ratio (OR) = 0.30; 95% confidence interval (CI): 0.15–0.61; P = 0.001]. Additionally, low high-density lipoprotein (HDL) levels were identified as independent negative predictors of super response (OR = 0.44; 95% CI: 0.24–0.81; P = 0.008).

Conclusion: Prior biologic therapy and HDL level were identified as the most important factors associated with super response.

Keywords: IL-17/IL-23 inhibitors, psoriasis, super response

Introduction

Psoriasis is a chronic, immune-mediated systemic inflammatory disease that affects approximately 2–3% of the population worldwide.[1] Recent studies have revealed that the T-cell-mediated immune response is central to the pathogenesis of psoriasis. Specifically, pathogenic T cells that produce high levels of interleukin (IL)-17 in response to IL-23 stimulation were shown to be the main drivers of the disease.[2]

Biologic agents developed based on this knowledge led to a significant paradigm shift in the treatment of psoriasis. Clinical trial data on monoclonal antibodies targeting the IL-17 signaling pathway (secukinumab, ixekizumab, and bimekizumab) and the newer IL-23p19 antagonists (tildrakizumab, guselkumab, and risankizumab) have provided further evidence that these cytokines are major triggers of the pathogenesis of psoriasis.[2,3] These findings confirm the central role of the IL-17/IL-23 axis in the pathogenesis of psoriasis and provide the scientific basis for focusing treatment strategies on this axis.

The elucidation of these different pathogenetic pathways has enabled the development of new targeted treatment approaches. The widespread and diverse use of biologics has

Submission: 5-Dec-2025
Acceptance: 25-Mar-2026 **Web Publication:** 08-Jun-2026

Access this article online
Quick Response Code:
Website: www.turkjdermatol.com
DOI: 10.4274/tjd.galenos.2026.42204

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How to cite this article: Ürün M, Güzelbaba MC, Gürsel Ürün Y. Evaluation of super response in plaque-type psoriasis patients treated with IL-17 and IL-23 inhibitors. Turk J Dermatol. 2026;20(2):64-71.

led to the emergence of new concepts in the literature, one of which is the term "super response".[3] Researchers have generally defined the concept of super response based on the initial response rate and the maintenance of that response over time.[4] However, there is no consensus on the definition of "super response" specific to psoriasis.[5] Table 1 summarizes the definition of "super response" for patients receiving IL-17 or IL-23 inhibitor therapy.[5-17]

The aim of the present study was to determine the sociodemographic and clinical characteristics of patients with psoriasis classified as non-super-responders (NSRs) and super responders (SRs) to IL-17 or IL-23 inhibitor treatment. A secondary objective was to determine the preferred treatment modalities for patients with SR status. Our findings are expected to contribute to the identification of biomarkers that predict clinical response in this group.

Materials and Methods

Study Design

This single-center, retrospective, cross-sectional, observational analysis included patients with moderate-to-severe plaque psoriasis who presented to the dermatology and venereal diseases outpatient clinic of Trakya University Faculty of Medicine between January 2020 and December 2024. Approval was obtained from the Ethics Committee of Trakya University (approval number: 15/11, date: 25.08.2025).

Inclusion Criteria

1) Age over 18 years,

2) Clinical and histopathological diagnosis of plaque psoriasis,

3) Use of IL-17 or IL-23 inhibitor therapy at the standard dose,

4) Regular follow-up for at least 28 weeks,

5) Informed consent.

Patients diagnosed with guttate, erythrodermic, or pustular psoriasis who were receiving IL-17 or IL-23 inhibitors in combination with any systemic therapy (acitretin, methotrexate, apremilast) or with ultraviolet therapy were excluded from the study.

Definition of Super Responder

In our study, super response was defined as a 100% improvement in psoriasis area and severity index (PASI) score (PASI 100) at week 16 that was maintained at week 28. To determine NSR and SR status, PASI scores at baseline (pre-treatment) and at 16 and 28 weeks after the start of treatment were extracted from patient records and evaluated. Patients who met the specified criteria were divided into the NSR and SR groups. The groups were compared in terms of gender, age, disease duration, comorbidities and psoriatic arthritis (PsA), smoking and alcohol use, body mass index (BMI), dermatological life quality index (DLQI), biologic agent used, number of prior systemic treatments, history of biologic use before the current treatment, drug continuity, drug regimen changes, and high-density lipoprotein (HDL), low-density lipoprotein (LDL), and total cholesterol values.

Table 1. Definitions of SR to IL-17 and IL-23 inhibitors in patients with psoriasis vulgaris

Authors	Publication date	Biologic agent	Definition of SR status
Morelli et al.[7]	December 2021	Secukinumab	Maintaining PASI 100 response up to weeks 88 and 100
Feldman et al.[6]	March 2022	Tildrakizumab	PASI 90 response at week 28
Reich et al.[8]	August 2022	Guselkumab	PASI 100 response at weeks 20 and 28
Ruiz-Villaverde et al.[9]	September 2022	Guselkumab	PASI 0 at weeks 12 and 24
Mastorino et al.[11]	December 2022	IL-17 and IL-23 inhibitors	In bio-naive patients, PASI 100 response at week 16 and maintained at week 100
Rompoti et al.[10]	January 2023	Brodalumab	PASI ≤ 1 at week 12 or week 16
Herranz-Pinto et al.[14]	July 2023	Guselkumab	PASI ≤ 2 after the third dose of guselkumab and PASI ≤ 1 maintained after subsequent doses for at least 52 weeks
Menéndez Sánchez et al.[15]	July 2023	IL-23 inhibitors	PASI 0 at week 16 and 24
Kim et al.[13]	October 2023	IL-17A, IL-23, TNF, and IL-12/23 inhibitors	PASI 100 response at weeks 48-52
Schäkel et al.[17]	October 2023	Guselkumab	PASI 100 response at weeks 20 and 28
Di Giulio et al.[5]	September 2025	IL-23 inhibitors	PASI ≤ 1 at week 16 and maintained at weeks 28 and 52
Zhang et al.[12]	October 2025	IL-17 inhibitors	PASI 0 before week 4 and maintained at week 24
Kojanova et al.[16]	October 2025	Guselkumab	PASI 0 at week 16 and 24

IL: Interleukin, PASI: Psoriasis area and severity index, TNF: Tumor necrosis factor, SR: Super-responder

The Charlson comorbidity index (CCI) estimates mortality risk for patients with various comorbidities. The prognosis is estimated based on 19 criteria, including age, with total scores ranging from 0 to 33.[18] In our study, CCI was assessed at the initiation of biologic therapy. Switching patterns between IL-17 and IL-23 inhibitors were classified into mutually exclusive categories, ensuring that each patient was included in only one treatment modification group.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics version 25. Non-parametric and categorical statistical methods were used to compare the sociodemographic and clinical characteristics of the NSR and SR groups. Because continuous variables did not meet the assumption of normality, comparisons of these variables (e.g., age and CCI score)were performed using the Mann-Whitney U test. The number of previous systemic treatments was also compared between groups using the Mann-Whitney U test, and the results were reported as Z values. Categorical variables, including gender, smoking and alcohol use, disease duration, comorbidity status, and baseline PASI and DLQI scores, were analyzed using the Pearson chi-square test or Fisher's exact test when expected cell counts were insufficient; Fisher's exact test was specifically used for comparisons involving ≥ 2 previously used biologic agents due to small cell counts. Logistic regression analysis was conducted to determine the effects of age, gender, baseline PASI score, prior biologic therapy, and HDL levels on the likelihood of achieving a super response. Results were expressed as odds ratios (ORs) with 95% confidence intervals (CIs). *P*-values less than 0.05 were considered statistically significant.

Results

A total of 200 patients were included in the study, 48% (n = 96) of whom were SRs. The mean age was 46.2 years, and males accounted for 54% of the sample. Over 70% of the patients had a disease duration longer than 10 years. There were no statistically significant differences in demographic factors, such as age, sex, smoking, and alcohol use, between the NSR and SR groups. Clinical characteristics such as disease duration, comorbidity, CCI, PsA, and BMI were also comparable between the groups ($P > 0.05$). A baseline PASI ≥ 10 was more common in the SR group ($P = 0.041$), while there was no difference in the frequency of baseline DLQI ≥ 10 between NSRs and SRs ($P > 0.05$) (Table 2).

Low HDL levels were significantly more common in the NSR group than in the SR group (51.0% vs. 33.3%, $P = 0.012$), whereas no significant between-group differences were observed for LDL ($P = 0.889$) or total cholesterol ($P = 0.472$) (Table 3).

Biologic treatment consisted of IL-17 inhibitors in 52% (n = 104) and IL-23 inhibitors in 48% (n = 96) of patients. Among biologic agents, the proportion of patients receiving guselkumab was numerically lower in super responders than in NSR (17.7% vs. 30.8%, $P = 0.032$). No statistically significant differences were observed for risankizumab ($P = 0.235$), ixekizumab ($P = 0.051$), or secukinumab ($P = 0.192$). Compared with the NSR group, SR patients were less likely to have received biologic therapy previously and more likely to have used only one biologic ($P = 0.002$ and $P = 0.007$, respectively). SR patients were more likely to continue receiving the same biologic agent, while NSR patients were more likely to switch drug regimens ($P = 0.019$) (Table 4).

Multivariate logistic regression analysis, including age, sex, baseline PASI score, prior biologic therapy use, and HDL levels, demonstrated that prior biologic therapy use was independently associated with a lower likelihood of achieving super response (OR = 0.30; 95% CI: 0.15–0.61; $P = 0.001$). Additionally, low HDL levels were identified as an independent negative predictor of super response (OR = 0.44; 95% CI: 0.24–0.81; $P = 0.008$). No significant associations were observed for age, sex, or baseline PASI score in the multivariate model ($P > 0.05$; Table 5).

Table 2. Sociodemographic and clinical characteristics of NSR and SR patients

	Non-super-responders (n = 104)	Super responders (n = 96)	Total (n = 200)	X^2/Z	*P**
Age, mean ± SD, range	47.48 ± 13.56 (20–76)	45.24 ± 13.55 (18–75)	46.32 ± 13.57 (18–76)	-1175.00	0.240
Sex, n (%)					
Male	51 (49)	57 (59.4)	108 (54)	2147.00	0.143
Female	53 (51)	39 (40.6)	92 (46)		
Smoking, n (%)					
Current-smoker	55 (52.9)	50 (52.1)	105 (52.5)	1201.00	0.549
Ex-smoker	17 (16.3)	21 (21.9)	38 (19)		
Never-smoker	32 (30.8)	25 (26)	57 (28.5)		

Table 2. Continued

	Non-super-responders (n = 104)	Super responders (n = 96)	Total (n = 200)	X^2/Z	*P**
Alcohol, n (%)					
Current drinker	28 (26.9)	20 (20.8)	48 (24)	2708.00	0.258
Social drinker	16 (15.4)	23 (24)	39 (19.5)		
Never-drinker	60 (57.7)	53 (55.2)	113 (56.5)		
Disease duration, n (%) (years)					
< 5	14 (13.5)	12 (12.5)	26 (13)	0.04	0.840
5-10	14 (13.5)	19 (19.8)	33 (16.5)	1452.00	0.228
> 10	76 (73.1)	65 (67.7)	141 (70.5)	0.69	0.406
BMI (kg/m²), n (%)					
Normal (< 25)	27 (26)	26 (27.1)	53 (26.5)	0.03	0.857
Overweight (25 to < 30)	44 (42.3)	32 (33.3)	76 (38)	1706.00	0.191
Obese (≥ 30)	38 (39.6)	33 (31.7)	71 (35.5)	1344.00	0.246
CCI, mean ± SD, range	1.38 ± 1.67 (0–7)	1.4 ± 1.48 (0–6)	1.04 ± 1.57 (0–7)	-0.645	0.519
PsA*, n (%)**	39 (37.5)	25 (26)	64 (32)	3012.00	0.083
Comorbidities**, n (%)**	39 (37.5)	42 (43.8)	81 (40.5)	0.81	0.368
Baseline PASI ≥ 10, n (%)	29 (27.9)	40 (41.7)	69 (34.5)	4196.00	0.041**
Baseline DLQI ≥ 10, n (%)	18 (17.3)	16 (16.7)	34 (17)	0.02	0.904

*Pearson chi-square, Fisher's exact test, Mann-Whitney U test
**Statistically significant
***Rheumatologist-diagnosed psoriatic arthritis
****Comorbidities included hypertension, diabetes, coronary artery disease, and non-alcoholic fatty liver disease
SD: Standard deviation, BMI: Body mass index, CCI: Charlson comorbidity index, PsA: Psoriatic arthritis, PASI: Psoriasis area and severity index, DLQI: Dermatological life quality index

Table 3. Lipid profiles of NSRs and SRs

	Non-super-responders (n = 104)	Super responders (n = 96)	Total	X^2	*P**
Low HDL (females < 50 mg/dL and males < 40 mg/dL)	53 (51)	32 (33.3)	85 (42.5)	6348.00	0.012**
High LDL (≥ 130 mg/dL)	40 (38.5)	36 (37.5)	76 (38)	0.02	0.889
Total cholesterol (≥ 200 mg/dL)	35 (33.7)	37 (38.5)	72 (36)	0.52	0.472

*Pearson chi-square
**Statistically significant
HDL: High-density lipoprotein, LDL: Low-density lipoprotein, NSRs: Non-super-responders, SRs: Super-responders

Table 4. Treatment characteristics of NSR and SR patients

	Non-super-responders (n = 104)	Super responders (n = 96)	Total (n = 200)	X^2/Z	*P**
Biologic agent used					
Guselkumab (IL-23 inh.)	32 (30.8)	17 (17.7)	49 (24.5)	4604.00	0.032**
Risankizumab (IL-23 inh.)	28 (26.9)	19 (19.8)	47 (23.5)	1412.00	0.235
Ixekizumab (IL-17 inh.)	21 (20.2)	31 (32.3)	52 (26)	3798.00	0.051
Secukinumab (IL-17 inh.)	23 (22.1)	29 (30.2)	52 (26)	1699.00	0.192
Number of previous systemic treatments***	1.71 ± 1.2 (1–6)	1.25 ± 0.82 (1–5)	1.49 ± 1.06 (1–6)	-2720.00	0.007**
Previous biologic agent use******	38 (36.5)	15 (15.6)	53 (26.5)	9.6	0.002**

Table 4. Continued

	Non-super-responders (n = 104)	Super responders (n = 96)	Total (n = 200)	X^2/Z	*P**
Number of previously used biologic agents***					
1	32 (30.8)	14 (14.6)	46 (23)	7385.00	0.007**
≥ 2	6 (5.8)	1 (1.04)	7 (3.5)	4208.00	0.067
Continuation of the same biologic agent	88 (84.6)	91 (94.8)	179 (89.5)	5501.00	0.019**
Switch from IL-17 inh. to IL-23 inh.	9 (8.7)	3 (3.1)	12 (6)	2706.00	0.100
Switch from IL-23 inh. to IL-17 inh.	7 (6.7)	2 (2.1)	9 (4.5)	2509.00	0.173

*Pearson chi-square, Fisher's exact test, Mann-Whitney U test
**Statistically significant
***Refers to all conventional systemic drugs received for psoriasis vulgaris and the total number of different drugs tried
****History of exposure to any biologic prior to the current biologic treatment; biologic-experienced patient
*****Number of biologic agents received prior to the current biologic therapy
IL: Interleukin, inh.: Inhibitor, NSR: Non-super-responder, SR: Super-responder

Table 5. Results of multivariate analysis of age, sex, baseline PASI, prior biologic use, and HDL as predictors of SR status

	B	OR (95% CI)	*P*-value*
Age	0.019	1.02 (0.996–1.043)	0.102
Sex	0.398	1.489 (0.818–2.707)	0.192
Baseline PASI***	0.482	1.62 (0.859–3.055)	0.136
Prior biologic therapy	-1.210	0.298 (0.147–0.606)	0.001**
Low HDL****	-0.826	0.438 (0.237–0.809)	0.008**

*Logistic regression analysis. The B value indicates the effect of each variable on log-odds (logarithmic probability)
**Statistically significant
***Baseline PASI: Patients with PASI ≥ 10 before initiation of biologic therapy
****Low HDL: Females < 50 mg/dL and males < 40 mg/dL
PASI: Psoriasis area and severity index, HDL: High-density lipoprotein, SR: Super-responder, OR: Odds ratio, CI: Confidence interval

Discussion

The most common form of psoriasis is plaque-type (psoriasis vulgaris), which affects both genders equally. Recent literature reports the age at onset in Türkiye as 44.5 years.[19,20] Although our study included a small sample of patients, their sociodemographic data were consistent with the literature.

Increasing evidence indicates that SR psoriasis patients may exhibit a distinct endotype with clinical features that differ from those of NSR patients.[21] Studying SRs helps us understand which patient subgroups derive the greatest benefit from biologic agents and implement personalized treatment strategies.[5,8] Despite advances in psoriasis treatment, it remains a clinical challenge because a subset of patients, unlike SRs, do not respond to biologic therapies.[21] Recent studies have reported that approximately 6.5% of psoriasis patients are treatment-resistant.[22]

The concept of super response was first used in the literature in 2019 by Talamonti et al.,[23] who found that carriers of the HLA-Cw6 allele showed a better treatment response to ustekinumab. A similar study conducted in HLA-Cw6-positive and -negative psoriasis patients receiving secukinumab showed that HLA-Cw6 allele positivity was associated with a better response.[7]

One of the most important studies on this subject is GUIDE, a phase IIIb study. The study evaluated SR patients who were treated with guselkumab and showed that the most decisive factors in achieving SR status were a disease duration longer than two years and a history of biologic therapy.[17] Higher SR rates have been reported in recent studies. Zhang et al.[12] reported an SR rate of 64.7%, while Di Giulio et al.[5] reported an SR rate of 63.8%. In our study, this rate was 48%. Higher rates may result from increased awareness of super response among clinicians.

There is no consensus on the effect of sex on SR status. Menéndez Sánchez et al.[15] reported that men were more prevalent among NSRs receiving IL-23 inhibitors, whereas Mason et al.[24] suggested that female sex was associated with NSR status. Although male dominance was noted in our SR group, the difference did not reach statistical significance.

Other factors affecting SR status include lower BMI, biologic naivety,[16] and comorbidities such as obesity, hypertension, and diabetes.[25] Ruiz-Villaverde et al.[9] reported that SR patients

were predominantly younger and had lower BMI, although they did not observe statistically significant differences. Similarly, our SR group was relatively young and had a lower BMI. This association may be related to a lower baseline inflammatory load in SR patients.

Disease duration has also gained relevance in the evaluation of SRs. For psoriasis, a disease duration of 2 years or less has been defined as short, and a disease duration of 2 years or more has been defined as long.[5] SR rates appear better in patients with shorter disease duration.[5,12] As net disease durations could not be determined in our study, these data could not be interpreted. In Türkiye, the use of biologic agents is permitted only after conventional treatment. For these reasons, it is challenging to identify psoriasis patients with a short disease duration and to evaluate the effectiveness of biologic treatment in the early stages of the disease.

In our study, no statistically significant association was found between disease duration and SR status. Furthermore, disease duration and biologic-naive status should be considered distinct clinical variables. This observation underscores the potential importance of timely initiation of biologic therapy, rather than relying solely on disease duration or biologic-naive status when making treatment decisions.[5,11-15]

Although PASI values indicate disease severity, it has been emphasized that PASI elevations may also be associated with recent markers of systemic inflammation.[26] The burden of systemic inflammation determines not only the severity of the disease but also the accompanying endothelial damage, the presence of metabolic syndrome, and the cardiovascular risk.[27] Previous studies on the relationship between high PASI and SR status have yielded contradictory results. In our study, there was a higher proportion of patients with baseline PASI $\geq$ 10 in the SR group. These findings are consistent with those of Mastorino et al.[11] and Liu et al.[21] found that baseline PASI did not differ significantly between NSR and SR patients. In another study, SR status was associated with low baseline PASI.[25] Although higher baseline PASI scores may indicate greater disease severity, biologic-naive status should nevertheless be considered an independent clinical variable, distinct from disease severity. Therefore, the higher super response rates observed in patients with elevated baseline PASI scores should not be interpreted solely as a consequence of biologic-naive status. Instead, baseline disease severity may represent a distinct clinical factor influencing treatment response. Additional studies are needed to clarify the relationship between PASI and super response.

In psoriasis, dyslipidemia results from chronic inflammation and increased levels of oxidized lipids in psoriatic skin.[28] Numerous studies have demonstrated lower HDL levels in patients with psoriasis than in healthy controls. In our study, patients with low HDL values were less likely to achieve SR status. This is similar to a study by Liu et.,[21] in which HDL was the most prominent predictor among all patients. Low HDL was associated with an 87% reduction in the likelihood of achieving a PASI 90 response at 52 weeks. In another study, failure of multiple biologic treatments was found to be more frequent among patients with psoriasis who had a history of hyperlipidemia.[29] The persistent systemic inflammatory environment in psoriasis patients may contribute to reduced treatment efficacy and partially explain these findings. However, further research is needed to confirm the relationship between HDL levels and SR.

When the effect of previous treatment on SR status was evaluated, we observed no difference between the groups with respect to the conventional systemic treatments received. This is consistent with earlier studies showing that conventional treatments received before starting biologic therapy do not affect SR status.[13,27] However, previous exposure to biologic treatments has an effect. Multi-treatment resistance in psoriasis is defined according to the number of discontinued biologic agents ($\geq$ 2 to $\geq$ 9) and biologic classes ($\geq$ 2, $\geq$ 3, or 4).[30] In our study, there was no statistically significant difference between NSR and SR patients in the proportion previously exposed to 2 or more biologic agents. However, this result should be interpreted with caution, given the small number of patients with a history of using two or more biologic agents.

Achieving a super response also affects treatment duration for patients with psoriasis. Although our study was limited to 28 weeks, consistent with other studies, a higher proportion of SR patients continued the same drug treatment compared with NSR patients.[11,21] Being familiar with the characteristics of SR patients and personalizing treatment are important for treatment continuity.

SR rates vary depending on the biologic agents used. One study reported that SRs were more likely to receive IL-17 inhibitor therapy, with ixekizumab and brodalumab showing the highest SR rates.[11] In a study evaluating SR to IL-23 inhibitors, the proportion of SRs was higher in the risankizumab group than in groups receiving other IL-23 inhibitors.[15] In another study, no difference in SR rates was observed among IL-23 inhibitors.[5] In our study, subgroup analyses showed comparable rates of achieving a super response among patients treated with IL-23 inhibitors (guselkumab, risankizumab) and those treated with IL-17 inhibitors (ixekizumab, secukinumab). The highest SR rate was observed among patients receiving ixekizumab, whereas the lowest was observed among those receiving guselkumab. A recent meta-analysis showed that the PASI 75 response rates were highest for bimekizumab, brodalumab, and ixekizumab. In the network meta-analysis,

the PASI 90 response rate was similar between IL-17 and IL-23 agents, with bimekizumab producing the fastest response.[31] In another study, the highest PASI 75 response rate at week 52 was observed with guselkumab; however, that study was also conducted retrospectively. In our study, the unequal distribution of patient subgroups receiving biological therapies makes it difficult to interpret these findings. Additionally, our data collection ended at Week 28, which limits our ability to evaluate long-term SR.

Study Limitations

Limitations of our study include its retrospective design and the small number of patients in the subgroups. Moreover, follow-up was limited to 28 weeks. Finally, certain sociodemographic (e.g., socioeconomic level) values, clinical (e.g., genital involvement) values, and laboratory values (e.g., C-reactive protein concentration, sedimentation rate) could not be evaluated due to missing data.

Conclusion

Sociodemographic characteristics did not appear to significantly influence SR status. Prior biologic therapy and low HDL levels were independently associated with a lower likelihood of achieving a super response, whereas baseline PASI score was not independently associated with a super response after multivariate adjustment. The finding that SRs are more likely to continue the same biologic agent is an important contribution to our understanding of the characteristics of the SR patient group.

Ethics

Ethics Committee Approval: Approval was obtained from the Ethics Committee of Trakya University (approval number: 15/11, date: 25.08.2025).

Informed Consent: Written informed consent was obtained from all patients before study initiation for participation and for the use of anonymized clinical data in scientific research.

Footnotes

Surgical and Medical Practices: M.Ü., M.C.G., Y.G.Ü., Concept: M.Ü., M.C.G., Y.G.Ü., Design: M.Ü., M.C.G., Y.G.Ü., Data Collection or Processing: M.Ü., M.C.G., Y.G.Ü., Analysis or Interpretation: M.Ü., M.C.G., Y.G.Ü., Literature Search: M.Ü., M.C.G., Y.G.Ü., Writing: M.Ü., M.C.G., Y.G.Ü.

Conflict of Interest: The authors declare that they have no conflict of interest.

Financial Disclosure: The authors declared that this study received no financial support.

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