Original Article

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Real-world Efficacy, Safety, and Drug Survival of IL-23 Inhibitors in Overweight and Obese Patients with Psoriasis: A 52-week Retrospective Study

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Abstract

Aim: Obesity is a common comorbidity in psoriasis and has been associated with reduced response to biologic therapies. However, data on the real-world effectiveness of interleukin (IL)-23 inhibitors in overweight and obese patients with psoriasis remain limited. This study aimed to evaluate the efficacy, safety, and drug survival of IL-23 inhibitors in this patient population over 52 weeks.

Methods: This retrospective, single-center observational study included patients with psoriasis and a body mass index (BMI) ≥25 kg/m^2 who were treated with guselkumab or risankizumab between November 2021 and November 2025. Efficacy was assessed using absolute Psoriasis Area and Severity Index (PASI) scores and PASI75/90/100 response rates at predefined time points. Drug survival, adverse events, and reasons for discontinuation were evaluated. Multivariable logistic regression analysis was performed to identify independent predictors of achieving PASI90 at week 52.

Results: Among the 75 included patients, the mean PASI score at baseline was 13.7±7.7, which improved to 0.8±1.1 at week 52. Psoriasis Area and Severity Index 90 and PASI100 response rates increased from 37.3% and 12.0% at weeks 12-16 to 77.5% and 56.3% at week 52, respectively. Overweight patients achieved significantly higher PASI90 response rates than obese patients at weeks 28 and 52. In multivariable analysis, lower BMI and biologic-naïve status remained independent predictors of achieving PASI90 at week 52. The 52-week drug survival rate was 94.7%. Treatment was discontinued in 4 patients (5.3%), most commonly because of secondary loss of response. The most common adverse events were infections. No cases of hepatitis B or tuberculosis reactivation were observed.

Conclusion: Interleukin-23 inhibitors demonstrated sustained clinical effectiveness, high 52-week drug survival, and a favorable safety profile in overweight and obese patients with psoriasis. Although lower BMI was independently associated with higher odds of achieving PASI90 by week 52, drug survival remained comparable across BMI groups, supporting the use of IL-23 inhibitors in clinical practice.

Keywords: Psoriasis, guselkumab, risankizumab, interleukin-23, body mass index, treatment outcome

Introduction

Psoriasis is a chronic inflammatory disease affecting approximately 2-3% of the global population and around 1% in Türkiye (1,2). Previously considered a skin-limited condition, it is now widely accepted as a systemic disorder with multiple comorbidities, including obesity, type 2 diabetes mellitus, dyslipidemia, atherosclerotic cardiovascular disease, and metabolic-associated liver disease (3,4). A 1.5 times higher risk of obesity is seen in the psoriasis population relative to control subjects, marking it as one of the most widespread concurrent conditions (5). Moreover, obesity is associated with greater disease severity and an increased prevalence of psoriatic arthritis (5,6).

Biologics have significantly improved the management of psoriasis by targeting key inflammatory pathways and providing high levels of efficacy and durable disease control (7). However, obesity may influence the pharmacokinetics of biologic agents by altering drug distribution and clearance, potentially leading to

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reduced treatment efficacy (8). While some biologics allow weight-based dosing, most are administered at fixed doses. Reduced treatment responses have been reported with tumor necrosis factor-α (TNF-α) inhibitors in obese patients (9,10). According to data derived from a large multicenter registry, obesity is associated with a 25% and 30% reduction in PASI75 and PASI90 response rates, respectively, among patients receiving TNF-α and interleukin (IL)-23/Th17-targeted biologics (11). Notably, this effect appeared more pronounced in patients treated with TNF-α and IL-17 inhibitors. In line with these findings, higher body mass index (BMI) has also been identified as a predictor of shorter drug survival, largely driven by reduced treatment effectiveness (12). Despite the high efficacy of guselkumab and risankizumab in the overall psoriasis population, data on their performance in overweight or obese patients remain limited. We hypothesized that IL-23 inhibitors would provide sustained clinical effectiveness, a favorable safety profile, and high drug survival in overweight and obese patients with psoriasis.

Based on this hypothesis, the present study aimed to evaluate the effectiveness, safety, and drug survival of IL-23 inhibitors in overweight and obese patients with psoriasis over 52 weeks of follow-up. By providing real-world data from this patient population, this study may contribute to optimizing treatment strategies and supporting evidence-based therapeutic decision-making in routine clinical practice.

Materials and Methods

Compliance With Ethical Standards

The study received approval from the University of Health Sciences Türkiye, Istanbul Haseki Training and Research Hospital Non-Interventional Clinical Research Ethics Committee (approval number: 106-2026, date: 22.04.2026) and was conducted in accordance with the principles of the Declaration of Helsinki. Informed consent was waived due to the retrospective design of the study. All data were anonymized, and no identifiable patient information was used.

Study Design

This retrospective, single-center observational study was conducted at a tertiary referral center with a dedicated psoriasis outpatient clinic. Medical records of patients treated with biologics from November 2021 to November 2025 were reviewed. The patient selection process is summarized in Figure 1. Adult patients (≥18 years) with plaque psoriasis who received IL-23 inhibitors (guselkumab or risankizumab) and were classified as overweight (BMI 25-29.9 kg/m^2) or obese (BMI ≥30 kg/m^2) were included. Patients with a BMI <25 kg/m^2, those treated with biologics other than IL-23 inhibitors, and those with incomplete baseline or follow-up data were excluded. The primary study outcomes were absolute PASI scores and PASI75, PASI90, and PASI100 response rates at weeks 4, 12-16, 28, and 52. Secondary outcomes included drug survival; treatment discontinuation rates and reasons for discontinuation; and the occurrence of adverse events.

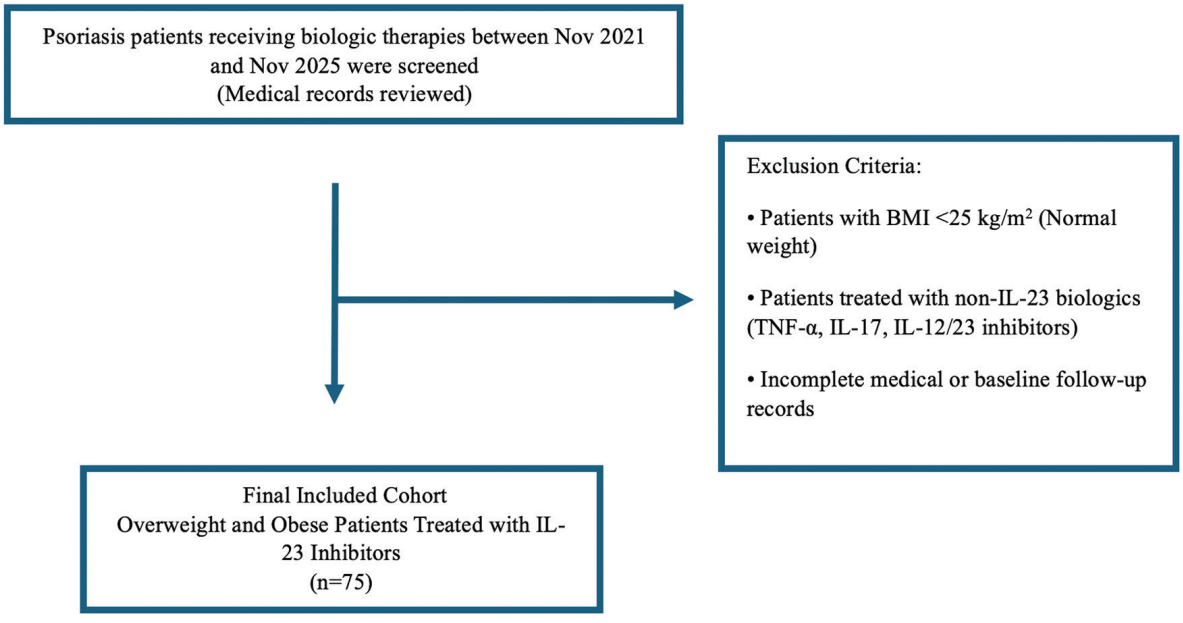

Figure 1. Flow diagram of patient selection and study cohort
BMI: Body mass index, IL: Interleukin, TNF-α: Tumor necrosis factor-α

Baseline demographic and clinical characteristics were collected, including age, sex, age at disease onset, family history of psoriasis, body weight, and BMI. In addition, data on psoriatic arthritis, involvement of special sites (scalp, palmoplantar, inverse, and nail psoriasis), and presence of systemic comorbidities were recorded.

Treatment-related data included prior conventional systemic therapies, prior biologic use, and prior phototherapy. Disease severity and therapeutic response were evaluated using the Psoriasis Area and Severity Index (PASI) at baseline and at weeks 4, 12-16, 28, and 52. Treatment responses were categorized as PASI75, PASI90, and PASI100, corresponding to ≥75%, ≥90%, and 100% improvement from baseline PASI scores, respectively.

Safety was assessed based on adverse events reported during routine clinical visits, physical examination findings, and laboratory monitoring. Hematological and biochemical analyses were performed every 3 months. Serology for hepatitis B, hepatitis C, and HIV was assessed at baseline and, in patients with negative baseline results, annually.

Patients with serological evidence of prior hepatitis B exposure [hepatitis B core antibody (anti-HBc) immunoglobulin G (IgG) positivity] underwent hepatitis B virus (HBV) deoxyribonucleic acid testing and were classified accordingly. These patients were co-managed by infectious disease or gastroenterology specialists, and laboratory monitoring was performed every 3 months.

Interferon gamma release assay (QuantiFERON-TB Gold) testing was performed at baseline and annually in patients with initially negative results. Chest radiography was performed at baseline and repeated at 6-month intervals during follow-up. Patients with positive QuantiFERON results were evaluated in collaboration with pulmonology specialists to rule out active tuberculosis and determine the need for latent tuberculosis infection (LTBI) prophylaxis.

For patients who discontinued treatment within the 52-week follow-up period, events leading to discontinuation were recorded. Primary cutaneous non-response was defined as failure to achieve a PASI75 response at weeks 12-16. Secondary cutaneous non-response was defined as loss of an initially achieved PASI75 response during follow-up. In patients with psoriatic arthritis, failure to achieve or maintain adequate control of joint symptoms was also considered treatment failure and classified as either primary joint non-response (within the first 12-16 weeks) or secondary joint non-response (loss of initially achieved disease control).

Statistical Analysis

Statistical analyses were conducted using IBM SPSS Statistics for Windows, version 26.0 (IBM Corp., Armonk, NY, USA). The normality of continuous variables was assessed using visual methods (histograms and Q-Q plots) and the Shapiro-Wilk test. Normally distributed continuous variables were presented as mean ± standard deviation, whereas non-normally distributed variables were presented as median (interquartile range). Categorical variables were presented as number (n) and percentage (%). Comparisons between two independent groups were performed using the independent samples t-test for normally distributed continuous variables and the Mann-Whitney U test for non-normally distributed continuous variables. Categorical variables were compared using the Pearson chi-square test or Fisher's exact test, as appropriate.

Drug survival was evaluated using Kaplan-Meier survival analysis, and differences between groups were assessed using the log-rank test. Univariable logistic regression analyses were performed to evaluate potential predictors of achieving PASI90 at week 52. Variables associated with the outcome in univariable analysis, together with clinically relevant potential confounders, were entered into the multivariable logistic regression model. Model calibration was assessed using the Hosmer-Lemeshow goodness-of-fit test, and model discrimination was evaluated using the area under the receiver operating characteristic curve (ROC-AUC). Odds ratios (ORs) with 95% confidence intervals (CIs) were reported. A two-sided p-value <0.05 was considered statistically significant.

Results

A total of 75 overweight or obese patients with psoriasis who were treated with IL-23 inhibitors were included in the study; 38 (50.7%) received guselkumab, and 37 (49.3%) received risankizumab. The most commonly involved special areas were the scalp (93.3%) and nails (88.0%). The most frequent comorbidities were hypertension (36.0%), hepatosteatosis (33.3%), dyslipidemia (32.0%), diabetes mellitus (25.3%), and coronary artery disease (22.7%). A total of 42.7% of patients had previously been treated with biologics, most commonly with ustekinumab (20.0%), secukinumab (16.0%), and ixekizumab (13.3%). Baseline demographics and clinical features are summarized in Table 1.

The mean baseline PASI score was 13.7±7.7 (Table 2). At week 4, the mean PASI score decreased to 3.6±3.3 and to 2.2±3.0 at weeks 12-16. PASI90 and PASI100 response rates increased progressively from 37.3% and 12.0% at weeks 12-16 to 71.6% and 44.6% at week 28, reaching 77.5% and 56.3% at week 52, respectively. Detailed response rates at all time points are presented in Table 2. Exploratory subgroup analyses are summarized in Supplementary Tables S1 and S2. Overweight patients demonstrated significantly higher PASI90 response rates at weeks 28 and 52 than those of obese patients, whereas no statistically significant differences were observed in

Table 1. Baseline demographic and clinical characteristics of patients	
	Total group (n = 75)
Gender, n (%): male/female	46 (61.3)/29 (38.7)
Age, years, mean ± SD	52.3±14.2
Age at disease onset, years, mean ± SD	32.1±15.3
Psoriasis in first-degree relatives, n (%)	25 (33.3)
Body mass index, mean ± SD	29.9±4.0
Biologic-experienced patients, n (%)	32 (42.7)
Baseline PASI, mean ± SD	13.7±7.7
Total treatment duration, months, mean ± SD	34.6±11.6
Psoriatic arthritis, n (%)	38 (50.7)
Involvement of special areas	**n (%)**
Scalp	70 (93.3)
Nail	66 (88.0)
Inverse	10 (13.3)
Palmoplantar	7 (9.3)
Comorbidities	**n (%)**
Hypertension	27 (36.0)
Hepatosteatosis	25 (33.3)
Dyslipidemia	24 (32.0)
Diabetes mellitus	19 (25.3)
Coronary artery disease	17 (22.7)
Thyroid disease	6 (8.0)
Psychiatric comorbidities	5 (6.7)
Cerebrovascular disease	1 (1.3)
Inflammatory bowel disease	1 (1.3)
Previous cutaneous malignancy	1 (1.3)
Previous non-cutaneous malignancy	0 (0.0)
Latent tuberculosis infection n (%)	30 (40.0)
Hepatitis B status	**n (%)**
Isolated anti-HBc IgG positivity	3 (4.0)
Resolved hepatitis B infection	13 (17.3)
Non-immune (susceptible)	45 (60.0)
Vaccinated	14 (18.7)
Previous systemic treatments	**n (%)**
Narrowband UVB	31 (41.3)
Methotrexate	58 (77.3)
Acitretin	33 (44.0)
Cyclosporine	15 (20.0)
Etanercept	5 (6.7)
Adalimumab	7 (9.3)
Infliximab	2 (2.7)
Certolizumab	3 (4.0)
Ustekinumab	15 (20.0)
Secukinumab	12 (16.0)
Ixekizumab	10 (13.3)
Guselkumab	2 (2.7)
Risankizumab	3 (4.0)
BMI: Body mass index, NB-UVB: Narrowband ultraviolet B, PASI: Psoriasis Area and Severity Index, SD: Standard deviation, y: Years, IgG: Immunoglobulin G	

Table 2. Treatment outcomes during IL-23 inhibitor therapy					
IL-23 inhibitor treatment			**n (%)**		
Guselkumab			38 (50.7)		
Risankizumab			37 (49.3)		
Total treatment duration, months, mean ± SD			34.6±11.6		
Time point	**n**	**Absolute PASI (mean ± SD)**	**PASI75 n (%)**	**PASI90 n (%)**	**PASI100 n (%)**
Baseline	75	13.7±7.7	-	-	-
Week 4	75	3.6±3.3	46 (61.3)	5 (6.7)	2 (2.7)
Week 12-16	75	2.2±3.0	71 (94.7)	28 (37.3)	9 (12.0)
Week 28	74	1.1±1.5	73 (98.6)	53 (71.6)	33 (44.6)
Week 52	71	0.8±1.1	68 (95.8)	55 (77.5)	40 (56.3)

PASI75/90/100 indicate ≥75%, ≥90%, and 100% improvement from baseline PASI score
PASI: Psoriasis Area and Severity Index, SD: Standard deviation

PASI75 or PASI100 responses between overweight and obese patients (Supplementary Table S1). Similarly, risankizumab-treated patients achieved higher PASI response rates at several early and intermediate time points, whereas no statistically significant differences were observed at week 52 (Supplementary Table S2). Univariable logistic regression analyses are presented in Supplementary Table S3. Lower BMI and biologic-naïve status were significantly associated with achieving PASI90 at week 52, whereas age, sex, psoriatic arthritis, and comorbidities were not. In multivariable logistic regression analysis (Table 3), increasing BMI (adjusted OR 0.773, 95% CI 0.640-0.933; p=0.007) and previous biologic exposure (adjusted OR 0.149, 95% CI 0.037-0.606; p=0.008) were independently associated with lower odds of achieving PASI90 at week 52 after adjustment for diabetes mellitus and hepatic steatosis. The model demonstrated acceptable discrimination (ROC-AUC=0.789) and good calibration, as assessed by the Hosmer-Lemeshow goodness-of-fit test (χ^2=6.303, df=8, p=0.613).

Hepatitis B infection was present in 21.3% (n=16) of patients (Table 1). All three patients with isolated anti-HBc IgG positivity received antiviral prophylaxis. Among the 13 patients with resolved hepatitis B infection, 10 received prophylaxis. No evidence of HBV reactivation was detected in any patient over the course of follow-up.

Latent tuberculosis infection was detected in 40.0% (n=30) of patients. All patients started prophylaxis; it was successfully completed in all but one, who discontinued treatment due to adverse effects. No cases of LTBI reactivation were observed.

The most commonly observed adverse events were infections, including upper respiratory tract infections (6.7%) and dermatophyte or Candida infections (2.7%) (Table 4). One case was newly diagnosed with basal cell carcinoma during follow-up.

Treatment was discontinued in 4 patients (5.3%) during the 52-week follow-up period (Table 4), primarily due to secondary non-response (2.7%), followed by primary non-response (1.3%) and adverse events (1.3%). The overall 52-week drug survival rate was 94.7% (Figure 2). Kaplan-Meier analysis showed no significant difference in drug survival between overweight and obese patients (log-rank p=0.143) or between patients treated with guselkumab and those treated with risankizumab (log-rank p=0.319).

Discussion

In this real-world study, IL-23 inhibitors demonstrated high and sustained efficacy in psoriasis patients with higher BMI. At weeks 12-16, PASI90 and PASI100 responses were attained in 37.3% and 12.0% of patients, respectively; these increased to 77.5% and 56.3% at week 52. In phase 3 clinical trials, both guselkumab and risankizumab have demonstrated high efficacy, with substantial PASI90 and PASI100 response rates as early as week 16 (13,14). Although response rates at earlier time points in the present study were lower than those reported in clinical trials, treatment outcomes at week 52 (PASI90: 77.5%; PASI100: 56.3%) were comparable to the efficacy rates

Table 3. Multivariable logistic regression analysis of predictors of achieving PASI90 at week 52		
Variable	**Adjusted OR (95% CI)**	**p**
BMI (continuous, kg/m^2)	0.773 (0.640-0.933)	0.007
Previous biologic exposure	0.149 (0.037-0.606)	0.008
Diabetes mellitus	1.516 (0.308-7.471)	0.609
Hepatosteatosis	0.610 (0.151-2.464)	0.487

Variables entered into the model: BMI (continuous, kg/m^2), previous biologic exposure, diabetes mellitus, and hepatic steatosis. The model demonstrated acceptable discrimination (area under the receiver operating characteristic curve [AUC]=0.789) and good calibration according to the Hosmer-Lemeshow goodness-of-fit test (χ^2=6.303, df=8, p=0.613).
BMI: Body mass index, CI: Confidence interval, OR: Odds ratio, PASI: Psoriasis Area and Severity Index

Table 4. Adverse events and reasons for treatment discontinuation	
Adverse events	**n (%)**
Upper respiratory tract infection	5 (6.7)
Urinary tract infection	1 (1.3)
Dermatophyte or Candida infection	2 (2.7)
Non-melanoma skin cancer	1 (1.3)
Hepatitis B reactivation n (%)	**0 (0.0)**
Tuberculosis reactivation n (%)	**0 (0.0)**
Reasons for treatment discontinuation	**n (%)**
Primary cutaneous non-response	1 (1.3)
Secondary cutaneous non-response	1 (1.3)
Secondary joint non-response	1 (1.3)
Non-melanoma skin cancer (basal cell carcinoma)	1 (1.3)

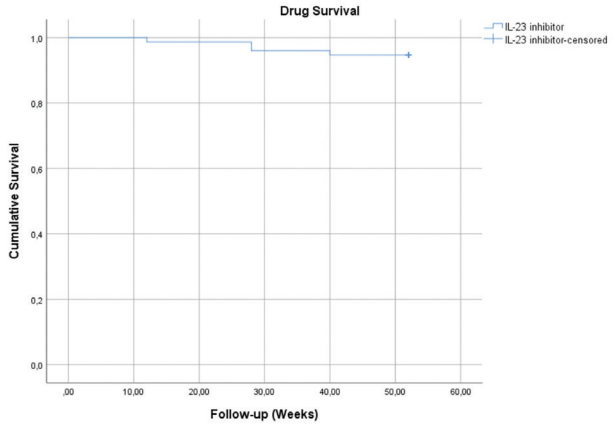

Figure 2. Kaplan-Meier curve showing the 52-week drug survival of the study cohort
IL: Interleukin

observed in clinical studies of both agents (13,14). It should be noted, however, that the mean baseline PASI score in our cohort (13.7) was considerably lower than those reported in the phase 3 trials of risankizumab (20.6) and guselkumab (21.9) (13,14). This likely reflects routine clinical practice in which biologic therapy may be initiated for patients with lower PASI scores owing to involvement of special areas, psoriatic arthritis, impaired quality of life, or previous treatment failure. In line with this, scalp and nail involvement were highly prevalent in our cohort, affecting 93.3% and 88.0% of patients, respectively. In addition, the lower baseline PASI may have partly contributed to the high PASI100 rates observed at week 52.

Pooled analyses of phase 3 studies of IL-23 inhibitors have shown that treatment responses are generally consistent across BMI categories, although slightly reduced responses have been reported in obese patients compared to individuals with lower BMI (13,15). These findings indicate that IL-23 inhibitors remain highly effective despite the potential adverse effects of obesity on treatment outcomes.

Existing real-world evidence regarding the influence of BMI on the effectiveness of IL-23 antagonists has yielded conflicting results. In a study assessing predictors of achieving super-responder status (complete clearance at week 20) with guselkumab, obesity was identified as a negative predictor, along with prior biologic experience and higher baseline PASI scores (16). Similarly, a real-world registry study demonstrated that although patients across all BMI categories experienced substantial improvements with guselkumab, obese patients were less likely to achieve relative endpoints such as PASI90 or complete clearance compared to those with lower BMI (17). Another study demonstrated reduced complete clearance rates at week 16 in obese patients compared to individuals with lower BMI; however, this difference attenuated over time, suggesting that obesity may be associated with a delayed response to risankizumab (18). Similarly, heavier patients have been shown to be less likely to achieve a PASI75 response at early time points, suggesting a delayed response to guselkumab.

In contrast, other real-world studies evaluating IL-23 inhibitors, including guselkumab, risankizumab, and tildrakizumab, have shown that overweight and obese patients can achieve comparable overall treatment responses, with mean PASI scores decreasing to low levels over time, suggesting that increased body weight may not significantly impair long-term efficacy (19-21).

Taken together, studies suggest that although obesity may be associated with a slower or less pronounced early response and a lower likelihood of achieving higher levels of skin clearance, IL-23 inhibitors appear to maintain robust and sustained efficacy in overweight and obese patients. Consistent with these observations, overweight patients in our cohort achieved significantly higher PASI90 response rates at weeks 28 and 52 than obese patients, whereas PASI75, PASI100, and drug survival were comparable between the two BMI groups. Furthermore, lower BMI remained independently associated with achieving PASI90 at week 52 after adjustment for previous biologic exposure, diabetes mellitus, and hepatosteatosis. This finding suggests that increasing BMI may independently reduce the likelihood of achieving PASI90, even after accounting for potential metabolic confounders such as diabetes mellitus and hepatosteatosis. Similarly, risankizumab-treated patients achieved higher PASI response rates at several early and intermediate time points than did guselkumab-treated patients, whereas no significant differences were observed between the groups at week 52 or in drug survival. These findings may suggest differences in the timing of clinical response

rather than long-term effectiveness; however, as this was an exploratory comparison without adjustment for potential baseline differences between treatment groups, no definitive conclusions can be drawn. Larger multicenter comparative studies are warranted to determine whether clinically meaningful differences exist between individual IL-23 inhibitors in overweight and obese patients.

In addition, IL-23 inhibitors demonstrated a favorable safety profile, consistent with previous reports (22-24). Infections were the most commonly observed adverse events. No adverse events led to treatment termination; the only exception was one case of newly diagnosed basal cell carcinoma. A considerable proportion of patients had concomitant hepatitis B infection (n=16, 21.3%). Prophylaxis rates were high, with 13 of 16 patients receiving antiviral therapy. None of the cases developed reactivation of the HBV during follow-up, supporting the safety of IL-23 inhibitors. These results align with earlier research indicating that the risk of HBV reactivation remains minimal during treatment with IL-23 inhibitors (25-27). Similarly, LTBI was common (n=30, 40.0%). Prophylaxis was initiated in all patients and completed in all but one due to adverse effects. None of the cases showed reactivation. In accordance with available clinical research and observational real-world data, these results highlight the favorable safety profile of IL-23 inhibitors regarding their low potential for reactivation (28-32).

Study Limitations

This was a single-center study with a relatively small sample size and a retrospective design, which constituted the main limitations. In addition, the absence of a normal-weight comparator group precludes direct comparisons across BMI categories and limits conclusions regarding the independent effect of BMI on treatment outcomes. However, the study provides valuable real-world clinical data on the effectiveness, safety, and drug survival of IL-23 inhibitors in an overweight and obese population with psoriasis, a group known to have a reduced response to treatment. Larger, multicenter comparative studies including patients from all BMI categories are warranted. Additionally, the small number of treatment discontinuations during follow-up limited the statistical power of the drug survival analyses and subgroup comparisons; therefore, these findings should be interpreted with caution. Despite these limitations, the present study provides valuable real-world evidence supporting the sustained effectiveness, favorable safety profile, and high drug survival of IL-23 inhibitors in overweight and obese patients with psoriasis.

Conclusion

IL-23 inhibitors demonstrated sustained clinical effectiveness, high 52-week drug survival, and a favorable safety profile in this real-world cohort of overweight and obese patients with psoriasis. Although increasing BMI was independently associated with a lower likelihood of achieving PASI90 at week 52, drug survival remained comparable across BMI groups. These findings support IL-23 inhibitors as an effective, well-tolerated treatment option for overweight and obese patients with psoriasis in routine clinical practice.

Ethics

Ethics Committee Approval: The study received approval from the University of Health Sciences Türkiye, Haseki Training and Research Hospital Non-Interventional Clinical Research Ethics Committee (approval number: 106-2026, date: 22.04.2026) and was conducted in accordance with the Declaration of Helsinki.

Informed Consent: Informed consent was waived due to the retrospective design of the study.

Authorship Contributions

Surgical and Medical Practices: G.P.U., Y.S., Concept: G.P.U., Design: G.P.U., Data Collection or Processing: G.P.U., Y.S., Analysis or Interpretation: G.P.U., Y.S., Literature Search: G.P.U., Y.S., Writing: G.P.U., Y.S.

Conflict of Interest: No conflicts of interest were declared by the authors.

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Declaration on the Use of Artificial Intelligence (AI): Artificial intelligence has been used to assist with "language correction".

Supplementary Tables Link: https://d2v96fxpocvxx.cloudfront.net/7a593d95-86ec-4d2b-8dd3-26b0d0b79ea4/content-images/67b21eb4-07d0-4ea2-9ddc-76b4d2beb3cb.pdf

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