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TITLE PAGE

Title

Predictors of 12-Month DAPSA Remission With Guselkumab in Psoriatic Arthritis in a Multicenter Real-World Study

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Conflict of interest

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Study conception and design: Conforti A, Lo Gullo A

Data collection: All co-authors.

Statistical analysis: Conforti A, Lo Gullo A

Critical revision and approval of final version: All authors.

All authors take full responsibility for the integrity and accuracy of the data and analyses presented in this work.

ABSTRACT

Background

Real-world evidence on 12-month outcomes of guselkumab (GUS) in psoriatic arthritis (PsA) remains limited. This multicenter observational study aimed to identify predictors of 12-month DAPSA remission (DAPSA <4) in patients with PsA treated with GUS. Secondary objectives were to assess remission rates and changes in disease activity at 6 and 12 months.

Methods

We screened all patients with PsA initiating GUS across 26 Italian rheumatology centers. Data collected included demographics, disease activity measured by DAPSA, and psoriasis (PsO) extent classified as 0%, <10%, 10–20%, or >20% body surface area. A multivariable logistic regression model restricted to patients with an evaluable 6-month assessment was used to identify predictors of 12-month DAPSA remission. Covariates included age, sex, smoking status, body mass index, disease duration, number of prior advanced therapies, axial involvement, and 6-month articular and/or cutaneous response. Articular response was defined as DAPSA remission (DAPSA <4), and cutaneous response as improvement by at least one PsO body surface area severity category. A two-sided p value <0.05 was considered statistically significant.

Results

Of 278 initiators, 199 were evaluable at 6 months. At month 6, 18 patients had a combined articular and cutaneous response, 9 had an articular-only response, 74 had a cutaneous-only response, and 98 had no response. In intention-to-treat analyses, DAPSA remission was achieved by 12% at 6 months and 20% at 12 months; corresponding per-protocol rates were 16% and 30%. Median DAPSA decreased from 27.0 at baseline to 11.9 at 6 months and 8.6 at 12 months. In multivariable analysis, combined response (OR 64.6, 95% CI 5.7–731.2), joint-only response (OR 16.9, 95% CI 4.4–65.2), and skin-only response (OR 2.5, 95% CI 1.04–6.2) were associated with 12-month DAPSA remission.

Conclusion

In routine practice, 6-month response status stratified the probability of 12-month DAPSA remission. Early articular remission and combined articular-cutaneous response showed the strongest associations, whereas skin-only improvement was a modest but statistically significant predictor and should not be interpreted as a strong determinant of later articular remission.

MAIN TEXT

Introduction

Psoriatic arthritis (PsA) is a chronic inflammatory disease involving multiple musculoskeletal domains, including peripheral joints, the spine, entheses, and tendons, and is commonly associated with skin and nail psoriasis (PsO) (1-4). In recent years, the therapeutic armamentarium for PsA has expanded with several biological disease-modifying antirheumatic drugs (DMARDs) that have demonstrated efficacy in various clinical settings (5), although a significant number of patients experience persistent symptoms, disease progression, or treatment-related problems (6,7,8).

The crucial role of interleukin-23 (IL-23) in the pathogenesis of PsA has become increasingly evident (8). Guselkumab (GUS) is a human monoclonal antibody administered subcutaneously that selectively blocks the p19 subunit of IL-23 and is effective in both PsO and PsA (8-13). In the DISCOVER programme, GUS demonstrated sustained efficacy across multiple disease domains, including low disease activity over longer follow-up and substantial resolution of enthesitis (10-13).

Real-world studies evaluating the effectiveness and safety of GUS have generally involved limited follow-up durations and relatively small cohorts (14-19). Therefore, there remains a need for studies evaluating the mid-term (12-month) effectiveness of guselkumab in large real-world cohorts.

The primary objective of this study was to identify predictors of 12-month DAPSA remission in patients with PsA treated with guselkumab, using 6-month response status as the main prognostic exposure. Secondary endpoints included the assessment of remission rates and disease activity at 6 and 12 months.

Materials and Methods

Study Design

This retrospective, observational study evaluated the real-world effectiveness of GUS in patients with PsA. The study was conducted in accordance with the Declaration of Helsinki and received approval from the Area Vasta Emilia Nord (AVEN) Ethics Committee (protocol 34713). Informed consent was waived by the Area Vasta Emilia Nord Ethics Committee due to the retrospective nature of the study.

Patients

Consecutive adult patients with psoriatic arthritis (PsA) treated at 26 Italian rheumatology centres who initiated guselkumab between October 2019 and December 2024 were screened, consistent with the multicentre cohort previously described by Becciolini et al. Patients met PsA classification criteria (e.g., CASPAR) and received guselkumab for rheumatologic indications. Patients receiving guselkumab exclusively for dermatologic indications were excluded. Baseline (T0) was defined as the clinical assessment closest to guselkumab initiation (9). Articular disease activity was assessed using the Disease Activity in Psoriatic Arthritis (DAPSA) score. Cutaneous involvement was approximated as a percentage of body surface area (BSA) and stratified as follows: 0%, <10%, 10-20%, and >20%. An articular response was defined as the achievement of DAPSA remission (DAPSA <4). A cutaneous response was defined as an improvement from a higher to a lower BSA stratification class. The predictor analysis for 12-month remission was conducted among patients with an evaluable 6-month assessment because 6-month response status was the key prognostic exposure; this design is conditional on treatment persistence to 6 months and is therefore interpreted with acknowledgement of potential selection bias. We defined the following analysis sets: (i) Intention-to-treat (ITT): all initiators (n=278); (ii) 6-month evaluable cohort: patients with a recorded 6-month assessment used to define the key prognostic exposure (n=199); and (iii) Per-protocol (PP): patients who remained on therapy and had an evaluable 12-month visit (n=164). Patient flow was as follows: 278 consecutive

PsA patients initiated guselkumab during the study period; 199 had an evaluable 6-month assessment and formed the primary predictor cohort; 164 patients remained on treatment and had an evaluable 12-month visit (per-protocol). Reasons for non-evaluable 6-month assessment included treatment discontinuation, loss to follow-up, and missing clinical assessment in routine care. Patient flow and analysis populations are illustrated in Supplementary Figure S1.

From October 2019 to December 2024, 278 patients with PsA initiating GUS were enrolled across multiple Italian centers (Supplementary Figure S1). Baseline characteristics of all initiators are summarized in Table 1: median age was 56 years [IQR 50–63], male prevalence was 32.2%, median baseline DAPSA was 27.0 [IQR 20.2–32.8], and baseline PsO involvement (body surface area >0%) was present in 69.4% of patients. Of the 278 initiators, 199 (71.6%) had an evaluable 6-month assessment and 164 had an evaluable 12-month visit. Patients without a 6-month assessment were not included in the predictor model because the key exposure was the 6-month trajectory; this conditional design is addressed in the limitations. Remission rates (along with rates of low, moderate, and high disease activity) at 6 and 12 months were calculated for both the entire cohort who received at least one dose of guselkumab (intention-to-treat analysis, ITT) and for those who completed the 12-month follow-up (per-protocol analysis). For patients who discontinued therapy before these timepoints, the last recorded DAPSA value was carried forward (last observation carried forward, LOCF). For ITT analyses, missing follow-up DAPSA values due to discontinuation were imputed using LOCF. LOCF may bias estimates if discontinuation is related to outcome (e.g., lack of efficacy or adverse events). Therefore, ITT results should be interpreted as conservative/assumption-dependent and are complemented by per-protocol estimates.

Statistical Analysis

Continuous variables are reported as median values with interquartile ranges (IQR); categorical variables are presented as percentages and counts. A multivariate logistic regression analysis was performed on the subjects completing the first six months of treatment to identify predictors of 12-month

remission. The model included the following covariates: age, sex, smoking status, body mass index (BMI), disease duration, number of prior advanced therapies, axial involvement, and the presence of a 6-month articular and/or cutaneous response. Covariates were pre-specified a priori based on clinical relevance and prior literature rather than selected by automated procedures or univariate screening. Multicollinearity was assessed using variance inflation factors (VIF). Overall model fit was summarized using McFadden's pseudo-R^2. McFadden pseudo-R^2 was reported as an index of model fit for logistic regression and should not be interpreted as variance explained in the same sense as linear regression. DAPSA values at baseline, 6, and 12 months were compared using the Wilcoxon signed-rank test (paired) for non-parametric data. The multivariable logistic regression was fitted in the 6-month completer cohort with complete data for all covariates (analysis N = 188). Collinearity among predictors (e.g., treatment line and disease duration) was assessed using variance inflation factors (VIFs) and pairwise correlations; VIF >5 was considered indicative of problematic collinearity. As a sensitivity analysis to address potential correlation within the 4-level response construct, we refit the model using two binary predictors (6-month cutaneous response: yes/no; 6-month articular remission: yes/no), with and without an interaction term. Sensitivity models using binary 6-month predictors yielded directionally consistent associations (data not shown).

All analyses were conducted using Jamovi software (version 2.6.2). Statistical significance was defined as a two-tailed p-value < 0.05. Baseline characteristics of patients with and without an evaluable 6-month assessment were compared using Mann-Whitney U tests for continuous variables and chi-square or Fisher's exact tests for categorical variables, as appropriate. Patients with missing smoking status were excluded from the multivariable model under a complete-case approach.

Outcomes

The primary outcome was 12-month DAPSA remission (DAPSA <4). Secondary outcomes included (i) remission rates at 6 and 12 months in ITT and per-protocol analyses, (ii) distribution of DAPSA disease activity categories over time (remission/LDA/MDA/HDA), and (iii) change in DAPSA and

its components from baseline to 6 and 12 months. Throughout, 'remission' refers to DAPSA remission (DAPSA <4) (articular/PsA remission), not cutaneous remission.

Results

From October 2019 to December 2024, a total of 278 patients with psoriatic arthritis initiating guselkumab were enrolled across multiple Italian centers. As previously reported by Becciolini (9), the cohort had a median age of 57 years [IQR 50-63] and a median baseline DAPSA of 26 [IQR 23-29]. Cutaneous involvement (BSA >0%) was present in 68% of patients. Of 278 initiators, 199 (72%) were evaluable at 6 months, and 164 had an evaluable 12-month visit. Patients without a 6-month assessment were not included in the predictor model because the key exposure was the 6-month trajectory; this conditional design is addressed in the limitations.

Baseline characteristics of the full cohort of guselkumab initiators (n=278) are reported in Table 1. Overall, 199/278 (72%) patients had an evaluable 6-month assessment and were included in the predictor analysis. At the 6-month timepoint, 18 patients (9.0%) had achieved both articular remission and cutaneous improvement, 9 (4.5%) had achieved articular remission alone, 74 (37.2%) had cutaneous improvement without articular remission, and 98 (49.2%) had neither articular nor cutaneous response (Figure 1). The majority of patients in remission at six months maintained this state at 12 months. Notably, 27% (20/74) of patients with only cutaneous improvement at 6 months achieved DAPSA remission (DAPSA <4) by 12 months, a rate more than double that observed in patients with neither articular nor cutaneous response (12%, 12/98; p=0.01) (Figure 1). Discontinuation/unevaluable follow-up may be related to lack of effectiveness, adverse events, or loss to follow-up, which could lead to overestimation of outcomes in analyses conditional on 6-month persistence. Accordingly, we present ITT estimates (with LOCF) alongside per-protocol results and interpret predictor findings within this completer-based framework.

Multivariable analysis confirmed that any clinical improvement at 6 months was associated with a

higher probability of achieving 12-month DAPSA remission. Compared with patients with no response, the odds of 12-month DAPSA remission were highest in those with a combined response (OR 64.6, 95% CI 5.7–731.2), followed by those with articular remission alone (OR 16.9, 95% CI 4.4–65.2) and cutaneous improvement alone (OR 2.5, 95% CI 1.04–6.2) (Table 2). Confidence intervals for the combined-response and joint-only categories were wide, reflecting small subgroup sizes (combined n=18; joint-only n=9) and limited event counts; these estimates should therefore be interpreted primarily for direction rather than precise magnitude. The multivariable model included 188 patients. Multicollinearity was minimal (VIF range 1.03–1.11), and model fit was modest (McFadden pseudo-R^2 = 0.191). Disease activity category distributions over time for the ITT and per-protocol populations are shown in Figure 2. Seventy-nine patients (28%) lacked an evaluable 6-month assessment. Because the predictor analysis was conditional on having an evaluable 6-month visit, estimates of 12-month remission from this model should be interpreted within this conditional framework; ITT and per-protocol analyses are presented to provide complementary population-level estimates. In the intention-to-treat analysis, the denominator was all treated patients (n=278). The per-protocol analysis included patients who remained on treatment and completed the 12-month visit. Among the 164 patients in the per-protocol cohort who completed 12 months of GUS therapy and had an evaluable 12-month assessment, remission rates were higher, at 16% and 30% at 6 and 12 months, respectively. In this per-protocol subgroup, the median baseline DAPSA score of 27.0 demonstrated a significant reduction to 11.9 after 6 months and to 8.6 after 12 months of treatment (Figure 3A). This improvement was driven by significant reductions in each component of the DAPSA score at both timepoints compared to baseline (Figure 3B). As a sensitivity analysis, we refit the model using binary predictors (6-month cutaneous response, yes/no; 6-month DAPSA remission, yes/no). Results were directionally consistent with the primary model, supporting robustness to alternative response coding.

Discussion

In this multicenter real-world cohort of 278 patients with psoriatic arthritis (PsA) initiating guselku-

mab, we observed progressive improvement in disease activity over 12 months, with higher proportions of patients achieving a stringent treatment target, DAPSA remission (DAPSA <4), between the 6- and 12-month assessments. In the intention-to-treat (ITT) framework, remission increased from 12% at month 6 to 20% at month 12, while in the per-protocol cohort it rose from 16% to 30%. These categorical changes were mirrored by continuous improvements in disease activity: median DAPSA decreased from 27.0 at baseline to 11.9 at 6 months and 8.6 at 12 months, with reductions across DAPSA components. Together, these findings support that guselkumab can deliver clinically meaningful disease control in routine practice over a 12-month horizon, while also highlighting that attainment of very stringent remission remains challenging in a heterogeneous real-world population.

A central contribution of this study is the trajectory-based prognostic signal at 6 months. We found that response status at the 6-month visit strongly stratified the likelihood of reaching DAPSA remission at 12 months. Compared with patients with no early response, the highest odds of 12-month remission were observed in those achieving combined articular remission plus cutaneous improvement at 6 months (OR 64.7), followed by those with articular remission alone (OR 16.9). Importantly, patients with cutaneous improvement alone at 6 months also showed a statistically significant association with later DAPSA remission, albeit with a modest effect size (OR 2.5). This pattern suggests that early control of joint inflammation is more strongly associated with later DAPSA remission, whereas isolated cutaneous improvement may precede later articular improvement in only a subset of patients. The descriptive transition analysis reinforces this interpretation: 27% (20/74) of patients with skin-only improvement at month 6 achieved DAPSA remission by month 12, compared with 12% (12/98) among patients with neither joint nor skin response (p=0.01). Clinically, these observations support the 6-month assessment as a practical decision point for treat-to-target strategies, while avoiding overinterpretation of skin-only improvement as a strong determinant of joint remission.

At the same time, it is essential to interpret the magnitude and precision of these regression estimates appropriately. The confidence intervals for the combined-response and joint-only categories were

very wide, reflecting small subgroup sizes and sparse event counts. Thus, while the direction of association is robust, early articular remission and combined response predict later DAPSA remission, the exact magnitude should not be considered precise. This concern is particularly relevant when response categories are mutually exclusive, and one or more categories contain relatively few individuals. In recognition of potential instability due to sparse cells and potential correlation structure within the 4-level response variable, we conducted a sensitivity analysis using binary predictors (6-month cutaneous response, yes/no; 6-month articular remission, yes/no), with and without an interaction term; the resulting associations were directionally consistent with the primary analysis. The model explained a modest proportion of outcome variability (McFadden pseudo-$R^2 = 0.191$), indicating that additional unmeasured factors likely contributed to remission outcomes. This supports the core conclusion, 6-month trajectory provides prognostic information for 12-month DAPSA remission and does not depend solely on the categorical encoding of early response.

The finding that skin-only improvement at 6 months is associated with later DAPSA remission should be framed carefully as a modest but meaningful prognostic signal. The observed odds ratio and its confidence interval suggest that skin-only improvement is not a strong determinant of later joint remission; rather, it may identify a subset of patients in whom articular benefit accrues with longer exposure. In clinical terms, a patient with early cutaneous improvement but persistent joint activity at 6 months may not be an automatic "failure," particularly when other domains show stabilization or when there are reasons to avoid premature switching. However, the modest effect size also implies that skin-only improvement alone is insufficient as a definitive decision rule; the full clinical context remains necessary, including baseline burden, patient priorities, extra-articular manifestations, and the feasibility of intensifying or optimizing management without changing the mechanism of action. These findings are broadly consistent with efficacy signals from pivotal randomized trials and subsequent real-world studies, while underscoring that absolute remission rates depend strongly on endpoint stringency and analytic framework. DISCOVER-1, DISCOVER-2, and COSMOS showed sustained improvements across joint and skin domains with GUS, whereas registry and cohort data from

CorEvitas, GISEA, and other Italian real-world studies have documented meaningful 6- to 12-month improvements and treatment persistence (10-13,17-19,22). However, cross-study comparisons must account for differences in prior biologic exposure, follow-up schedules, and outcome definitions.

DAPSA remission is a stringent target and may yield lower absolute remission rates than other commonly used PsA targets. While DAPSA primarily reflects peripheral joint activity, it incorporates key patient-reported components (pain and patient global) and tender/swollen joint counts that overlap conceptually with elements used in composite treat-to-target outcomes such as minimal disease activity (MDA) and very low disease activity (VLDA). The congruence between DAPSA-based disease control and broader composite targets has been discussed in the literature, supporting DAPSA as a pragmatic tool in real-world monitoring (34). Nonetheless, because DAPSA does not directly incorporate certain PsA domains (e.g., enthesitis, dactylitis, axial symptoms, and skin in the same composite), using DAPSA remission as the primary endpoint should be understood as a focused assessment of articular/PsA remission in the peripheral joint domain, rather than comprehensive multidomain remission.

Real-world data also reinforce the concept that early improvements can consolidate with continued therapy. In the CorEvitas PsA/SpA Registry, “on-label persisters” treated with guselkumab showed clinically meaningful improvements at 6 months in cDAPSA, physician global assessments, pain, and skin burden, alongside approximately 79% persistence at 6 months (18). A companion CorEvitas analysis focused on patient-reported outcomes similarly documented meaningful improvements by 6 months (17). These observations align with our finding that response status at 6 months carries prognostic information for subsequent outcomes and that persistence in the setting of early improvement is often associated with improved control over time. Italian real-world evidence offers further context. In GISEA, Atzeni and colleagues reported relevant 6-month response rates (including MDA and DAPSA <14) and durability to 12 months, with a 76% retention rate; bDMARD-naïve status predicted 12-month MDA (OR 7.9) (19). While our primary endpoint was DAPSA remission (DAPSA <4), our ITT (20%) and per-protocol (30%) 12-month remission proportions are within an expected

range when considering endpoint stringency and real-world heterogeneity. Ruscitti and colleagues have also reported meaningful short-term improvements in real-world PsA cohorts treated with guselkumab, broadly consistent with trial benchmarks (16). Across these datasets, a coherent pattern emerges: guselkumab provides multidomain benefit, and outcomes may improve further over time among patients who remain on therapy, though the absolute rate of stringent remission is influenced by baseline characteristics, prior treatment history, and the outcome definition used.

The biological plausibility of a delayed articular response following early cutaneous improvement can be considered within the skin–enthesis–synovium framework of the IL-23/IL-17 axis. Experimental evidence indicates that IL-23 can act on entheseal resident immune cells, inducing cytokines and chemokines that drive inflammation and tissue remodeling (24). Mechanical stress and innate immune signals may amplify local entheseal inflammation in susceptible individuals (25). IL-23 signaling sits upstream of effector pathways that include IL-17 and other mediators, and the downstream responses may differ across tissues, depending on local cell composition and molecular pathotypes (25–27). In paired tissue studies, IL-23 pathway expression has been reported as stronger and more homogeneous in psoriatic skin than in synovial tissue, where expression can be heterogeneous and related to specific inflammatory pathotypes (28). This divergence provides a rationale for why IL-23 p19 blockade could yield relatively rapid skin improvement while joint responses may vary in onset across individuals. Emerging evidence from single-cell and spatial transcriptomic studies indicates shared immune features across skin and joint compartments in PsA, including shared CD8+ tissue-resident memory T-cell clones, while also highlighting differences in inflammatory signatures between tissues (29). Related single-cell work has shown clonal expansions of pro-inflammatory synovial CD8 T cells with tissue-homing receptor expression, supporting the concept of compartmentalized yet interconnected immune processes (30). Moreover, certain IL-17–producing populations may be partly IL-23 independent, which could contribute to variability in joint response kinetics despite IL-23 blockade and may be relevant to axial phenotypes (31-35). Within this mechanistic framework, a subset of patients could experience early suppression of cutaneous inflammation as an on-target

signal of pathway inhibition, with subsequent joint improvement accruing later as downstream inflammatory programs attenuate. This provides a plausible explanation for the modest but significant association between skin-only improvement at 6 months and later DAPSA remission, without implying that skin improvement alone is sufficient to predict joint outcomes with high certainty.

Several limitations require explicit emphasis. First, the predictor analysis is conditional on the availability of a 6-month assessment because the 6-month trajectory is the key prognostic exposure. If early non-evaluability was related to poor response, intolerance, adverse events, or loss to follow-up, the regression-based probabilities may overstate the likelihood of 12-month remission among all initiators. Second, ITT analyses relied on last observation carried forward (LOCF) to handle discontinuation-related missingness. LOCF assumes stability of outcome after dropout and may introduce bias if discontinuation is related to disease activity or response; accordingly, ITT results should be interpreted cautiously and alongside per-protocol estimates. Third, the mutually exclusive response strata generated sparse cells for some categories, contributing to wide confidence intervals and limiting precision in effect size estimation; we addressed this in part through sensitivity analyses using binary response predictors. The observed remission rates may appear lower than those reported in clinical trials; however, this is expected given the use of a stringent endpoint (DAPSA remission <4), the real-world setting, and a population with multiple prior treatment exposures. Less stringent targets typically yield higher response rates in similar cohorts. Additional limitations include the retrospective design, potential center-level variation in assessment timing and documentation, residual confounding typical of observational studies, and the use of BSA severity bands rather than PASI for skin assessment, which may reduce the granularity of cutaneous severity measurement. Finally, cohort characteristics that may influence observed responses, such as female predominance and the prevalence of fibromyalgia, could affect patient-reported components of DAPSA and related clinical assessments, potentially lowering the likelihood of meeting stringent remission thresholds despite improvement in inflammatory activity. In contrast to studies that evaluated all initiators from baseline, the present predictor model was conditional on having an evaluable 6-month visit because the

6-month response pattern was the exposure of interest; this difference should be considered when comparing prognostic findings across studies.

Despite these limitations, this study provides a pragmatic framework for clinicians: evaluating trajectory at 6 months, particularly evidence of articular remission and/or multidomain improvement, offers useful prognostic information regarding the probability of stringent DAPSA remission by 12 months. Importantly, the results support a nuanced approach for partial responders: while early articular remission and combined response have the strongest associations with later remission, skin-only improvement may still represent a favorable, modest prognostic signal for a subset, and could argue against automatic discontinuation at the 6-month mark when no other clinical concerns prompt a switch. Future prospective studies with standardized follow-up schedules, harmonized skin and joint metrics, and broader domain capture (including enthesitis and axial assessments) will be important to validate and refine trajectory-based decision rules and to clarify which patients are most likely to demonstrate delayed articular benefit under IL-23 p19 blockade.

Conclusion

Guselkumab was associated with improvement in disease activity over 12 months in this multicenter real-world PsA cohort, although stringent DAPSA remission was achieved in a minority of patients. Six-month response status helped stratify the probability of 12-month DAPSA remission: early articular remission and combined articular-cutaneous response showed the strongest associations, whereas skin-only improvement was a modest but statistically significant predictor. It should not be interpreted as a strong determinant of later articular remission. These findings support the value of reassessing patients at 6 months in routine care. However, the predictor analysis was conditional on 6-month evaluability, ITT analyses relied on LOCF, and some response strata were small, so effect estimates should be interpreted cautiously. Prospective validation is needed before these response patterns are used as stand-alone decision rules.

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TABLES and FIGURES

Table 1: Baseline characteristics of all guselkumab initiators (ITT cohort), n=278

Characteristic		Value
Male prevalence, %		32.2
Age, median (IQR), years		56 (50-63)
Smokers, %	Current Former Never Unknown	70.9 11.1 17.6 0.5
BMI, median (IQR), kg/m^2		27 (23-29)
PsA duration, median (IQR), months		80 (41-127)
mRDCI, median (IQR)		1 (0-3)
Inflammatory bowel disease prevalence, %		4.0
Fibromyalgia prevalence, %		21.6
Swollen joint count, median (IQR)		7 (4-10)
Tender joint count, median (IQR)		3 (1-5)
C-reactive protein, median (IQR), mg/dl		0.9 (0.3-3.0)
10-point visual analogue scale pain, median (IQR)		8 (7-8)
Patient global assessment, median (IQR)		7 (5-8)
DAPSA, median (IQR)		27.0 (20.2-32.8)
PsO body surface area involvement, %	0 <10% 10-20% >20% unknown	30.2 37.7 18.1 13.6 0.5
Axial subset prevalence, %		37.7
Line of treatment, median (IQR)		3 (2-4)
Concomitant conventional synthetic DMARD, %		30.7
Concomitant corticosteroids, %		31.2

Table 2

Multivariable Logistic Regression Analysis of Predictors of 12-Month DAPSA Remission Among Patients With an Evaluable 6-Month Response

Predictor		Odds ratio (OR)	95%CI	p-value
Age (per year)		1.0	1.0-1.0	p>0.05
Sex Male vs Female		1.5	0.6-3.4	p>0.05
Disease duration (per month)		1.0	1.0-1.0	p>0.05
BMI (per kg/m²)		1.0	0.9-1.1	p>0.05
Treatment line (per prior advanced therapy)		1.1	0.8-1.4	p>0.05
Axial PsA Presence vs Absence		0.8	0.4-1.9	p>0.05
6-month response (reference = "None")	Skin + joint	64.6	5.7-731.2	<0.001
	Skin only	2.5	1.04-6.2	0.04
	Joint only	16.9	4.4-65.2	<0.001
Smoking (reference= Never)	Former	0.7	0.2-2.0	p>0.05
	Current	1.3	0.4-4.1	p>0.05
Patients with missing smoking status were excluded from the multivariable model under a complete-case approach. The model was fitted on complete cases (N=188). Multicollinearity was low (VIF range 1.03–1.11), and McFadden pseudo-R^2 was 0.191.				

Figure 1

Proportion of patients achieving 12-month DAPSA remission (DAPSA <4) according to 6-month response category among patients with an evaluable 6-month assessment (n=199). Response categories were defined as articular remission and/or improvement in PsO body surface area severity class.

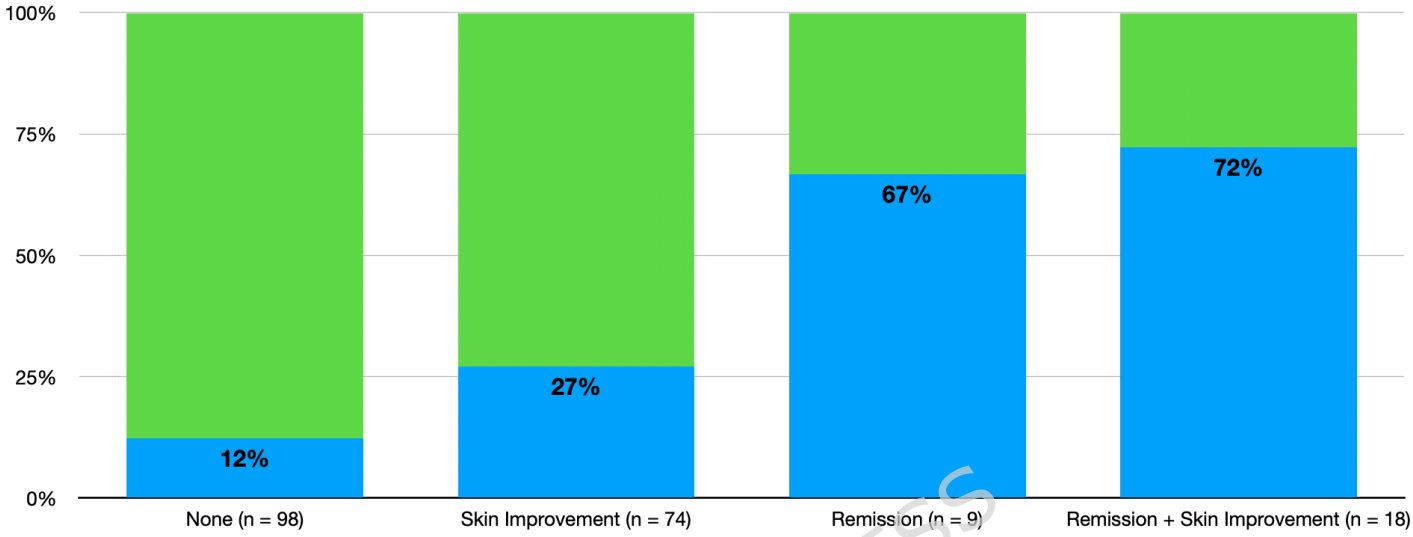

Figure 2

Figure 2. Distribution of DAPSA disease activity categories at baseline (T0), 6 months (T1), and 12 months (T2) in the ITT and per-protocol cohorts. The ITT cohort included all initiators (n=278), with missing post-baseline DAPSA values imputed using LOCF. The per-protocol cohort included patients who remained on therapy and had an evaluable 12-month visit (n=164). Sample sizes at each timepoint are indicated in the figure.

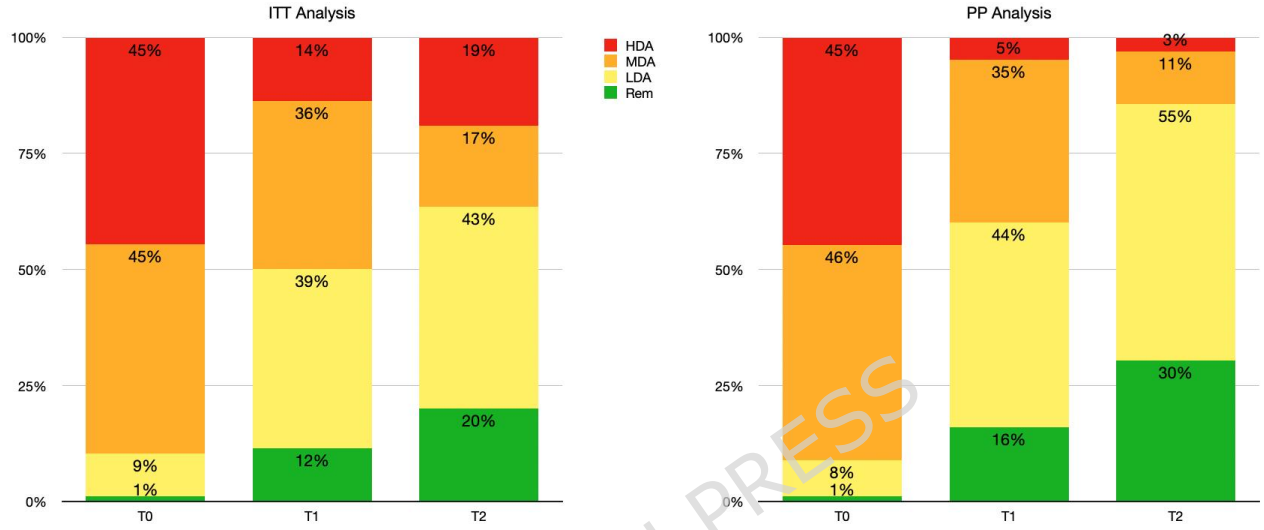

Figure 3

Figure 3. Change from baseline (T0) to 6 months (T1) and 12 months (T2) in the per-protocol cohort (n=164): (A) DAPSA, (B) tender joint count, (C) swollen joint count, (D) CRP, (E) pain VAS, and (F) patient global assessment. Sample sizes at each timepoint are indicated in the figure. Box plots show medians and interquartile ranges; whiskers indicate 1.5 × IQR.