Dermatology Practical & Conceptual

Tildrakizumab as a Potential Option for Early Psoriatic Arthritis in Patients with Metabolic Comorbidities and Psoriasis: a Case Series

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Key words: Tildrakizumab, Psoriatic arthritis, Metabolic syndrome, Psoriasis, Nail psoriasis

Citation: Artosi F, Lanna C, Gaeta Shumak R. Tildrakizumab as a Potential Option for Early Psoriatic Arthritis in Patients with Metabolic Comorbidities and Psoriasis: a Case Series. *Dermatol Pract Concept*. 2025;15(4):5570. DOI: https://doi.org/10.5826/dpc.1504a5570

Accepted: June 12, 2025; **Published:** October 2025

Funding: None.

Competing Interests: None.

Authorship: All authors have contributed significantly to this publication.

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ABSTRACT

Introduction: Psoriasis is a chronic relapsing inflammatory disease. and approximately 20% of psoriasis patients also develop psoriatic arthritis (PsA).. Interleukin 23 (IL-23) plays a crucial role in maintaining the T-helper (Th) 17 cell population derived from naïve Th1 cells as well as in sustaining inflammation at the enthesis and joints, acting as a pathogenic effector in PsA. Among anti-IL-23 monoclonal antibodies, ustekinumab, guselkumab, and risankizumab are approved in Europe for both psoriasis and PsA for psoriasis and PsA treatment. Tildrakizumab, another IL-23p19 inhibitor, is currently approved only for plaque psoriasis but has shown favorable safety in patients with metabolic comorbidities and potential efficacy in PsA.

Objectives: To retrospectively assess the efficacy of tildrakizumab on psoriatic arthritis manifestations in patients treated according to real-world clinical practice.

Methods: We conducted a retrospective analysis of eight patients affected by psoriasis, early PsA, and metabolic comorbidities, treated with tildrakizumab 100 mg every 12 weeks after induction. Descriptive and inferential statistical analyses were performed. Efficacy and safety were evaluated using standard clinimetric indices over a 28-week follow-up period.

Results: After 28 weeks, a significant mean reduction was observed in Psoriasis Area and Severity Index (-81.3%, P<0.001), Pain Visual Analogue Scale (-85%, P<0.002), Nail Psoriasis Severity Index

(-78%, P<0.002), Dermatology Life Quality Index (-86%, P<0.001), Physician Global Assessment (-73%, P<0.0003), and Disease Activity Index for PsA (-82%, P<0.000023). No adverse events were reported.
Conclusions: Tildrakizumab confirmed its efficacy in reducing signs and symptoms of early PsA, with high safety profile in our psoriasis patients also affected by multiple metabolic comorbidities.

Introduction

Psoriasis is a chronic relapsing inflammatory disease mainly involving the skin. The pathogenesis has not yet been fully understood and includes both immunological cells such as T lymphocytes but also epithelial cells such keratinocytes [1]. Physical, immunological, and infectious factors activate keratinocytes, favoring the release of antimicrobial peptides, DNA, and RNA self recruiting plasmacytoid dendritic cells in the affected skin. After the release of the interferon-α (INFa) by the plasmacytoid dendritic cells (pDC) and the subsequent activation of the dermal dendritic cells (dDCs), the acquired immunity is activated through IL-12 and IL-23. The subsequent differentiation of the T-helper (Th)-naive into Th1, Th17, and Th22 sets the groundwork of psoriatic plaque [1–3]. Keratinocytes contribute to maintaining the inflammatory process over time by promoting angiogenesis and releasing cytokines and chemokines for recruitment of macrophages, neutrophils, and Th17 lymphocytes[1].

Interleukin-23 (IL-23) plays a crucial role in psoriasis; produced by both keratinocytes and dendritic cells, it guarantees the maintenance, expansion, and activation of Th-17 lymphocytes, promotes epidermal hyperplasia, inhibits the function of regulatory T cells, and recalls neutrophils in psoriatic lesional skin with IL-17[2,4].

It is estimated that about 20% of patients with psoriasis are also affected by psoriatic arthritis (PsA), with an annual incidence of about 2% per year[2].

Differently from rheumatoid arthritis (RA), there is no evidence that IL-23 is increased in the synovial fluid in PsA [5]. Histological and molecular studies have shown that IL-23 is expressed within the RA synovium and plays a pro-inflammatory role, inducing both joint inflammation and bone destruction. However, its expression seems to be restricted to highly-inflamed synovial tissue rich in lymphoid aggregates[6]. On the other hand, as for IL-17, a higher concentration in the synovial fluid was found mainly in patients suffering from PsA [7]. Anti-IL-17 antibodies in PsA therapy play a key role but have a different safety profile compared to anti-IL-23 antibodies [8,9].

In order to explain the effectiveness of anti-IL-23 drugs on the arthritic process, some authors have suggested that elevated levels of IL-23 in the synovial fluid of PsA patients may play a key role in the pathogenesis[2, 9–11].

Currently available data support a clear association between nail psoriasis and PsA, with further evidence suggesting that improvement in nail disease correlates with improvement in joint involvement[13]. There is evidence that there is an anatomical continuity between the nail matrix and the entheses of the distal interphalangeal joints extensors, i.e., the most frequently affected site by PsA, confirming that both nail psoriasis and PsA could be considered two different aspects of the same pathogenetic process [14–16].

Currently, three monoclonal antibodies directed against IL-23 (or IL-12 and IL-23) have been approved for the treatment of both psoriasis and PsA, including guselkumab, ustekinumab, and risankizumab[17]. Tildrakizumab, another IL23 inhibitor, approved for plaque psoriasis treatment, is awaiting final approval for PSA. Indeed, phase 2 randomized clinical trials highlight, in patients suffering from PSA, a significant improvement in joint involvement except for dactylitis and enthesitis despite the mean duration of PsA was more than 6 years [18].

Psoriasis, as a systemic inflammatory disease, is closely intertwined with several comorbidities characterized by dysregulated systemic inflammation, with reciprocal influences that contribute to a vicious cycle whose management remains challenging[19]. In this context, it should be emphasized that psoriasis is associated with diabetes mellitus, high abdominal circumference, obesity, hypertension, and insulin resistance favoring progression of metabolic syndrome [19–21]. The therapeutic management of patients with metabolic syndrome requires drugs with high safety profile in terms of blood chemistry parameters and the other co-medications allowed in this class of patients [22,23].

As documented in RCTs, IL-23 inhibitors are a preferred option due to their renowned higher efficacy and better safety profile for psoriatic patients suffering from metabolic syndrome [19,21,23,24]. In a post hoc analysis of the ReSURFACE 1 and ReSURFACE 2 randomized trials, among patients with metabolic syndrome treated with tildrakizumab 100 mg, mean reductions were observed in fasting glucose, triglyceride levels, and systolic blood pressure compared to patients without metabolic syndrome. Similarly, those receiving the 200 mg dose experienced numerical decreases in both systolic and diastolic blood pressure compared to patients

without metabolic syndrome. Notably, patients without metabolic syndrome did not exhibit any increase in metabolic risk factors over the course of the study[23].

According to European guidelines, IL-23 or IL-17 inhibitors should be preferred in all psoriatic patients without contraindications to systemic agents or when they are ineffective [24]. Apremilast is considered a second-line treatment when biological therapies are contraindicated, and in this context, it should be favored over other agents in patients with metabolic comorbidities as it has demonstrated significant benefits in this population [22,24,25].

A case series of patients affected by moderate-to-severe psoriasis, early PsA, and metabolic disorders treated with tildrakizumab was carried out. After 28 weeks of treatment, patients achieved complete or partial resolution of both psoriasis and articular disease.

Methods

We enrolled eight patients with moderate-to-severe psoriasis and early PsA who were treated at the Dermatology Unit of Tor Vergata University Hospital with tildrakizumab 100 mg at baseline (T0), after four weeks, and with a maintenance dose of 100 mg every 12 weeks. All patients signed the informed consent for clinical data collection.

Exclusion criteria were patients <18 years old and those who refused to sign the informed consent for the collection of clinical data.

The following data were collected for each patient at baseline and after 28 weeks of follow-up: demographic data, psoriasis area and severity index (PASI), Pain Visual Analogue Scale (PAIN VAS) score, Nail Psoriasis Severity Index (NAPSI), the Patient Global Assessment (PGA), the Disease Activity in Psoriatic Arthritis (DAPSA) [26], and the Dermatology Life Quality Index (DLQI) [27].

Data on previous treatments for psoriatic and/or arthritic disease, comorbidities, and related drugs were also collected.

The diagnosis of PsA followed the CASPAR classification criteria for PsA [28,29].

The presence of PsA was suspected in each patient with a positive anamnesis of specific signs and symptoms such as morning stiffness lasting more than 30 minutes, joint tenderness, pain worse after rest and relieved by exercise, current or reported presence of swelling joint, dactylitis, limited joint mobility, and joint deformity [17,21]. All patients had a confirmatory color Doppler ultrasound and/or magnetic resonance imaging of the hands and/or feet to evaluate the presence of synovitis, dactylitis, tenosynovitis, erosions, bone edema, and/or enthesitis [29].

All patients received tildrakizumab therapy administered with 100 mg subcutaneous injections, with a dosage established according to the technical data sheet (induction and maintenance) for at least 28 weeks [23].

Statistical Analysis

Descriptive and inferential analyses were performed. The results are expressed as means or percentages, considering the type of each analyzed variable. To analyze the differences in terms of PASI, DLQI, and other indexes or parameters of interest before and after treatment at different timepoints (weeks), the t-test or ANOVA was used, followed by the Bonferroni correction ad hoc test. Differences of $P<0.05$ were considered statistically significant. All statistical analyses were performed using the SPSS 20.0 statistical package (SPSS Incorporated, Chicago)[21].

Results

Eight patients affected by psoriasis and early PsA were enrolled to start Tildrakizumab 100 mg treatment in the period between March 2022 and December 2023. The patients' demographics and clinical data are reported in Table 1. The mean duration of PsA at baseline was less than one year. No patient had PsA with axial involvement.

Five patients (62.5%) were male, and the mean age was 56 years. The mean PASI at T0 was 14.13. In six patients CRP values were found to be beyond the normal upper limit for the laboratory cutoff (1 mg/dl). The mean baseline PAIN VAS was ≃50.

Five (62.5%) patients had nail involvement, with a mean hand NAPSI score of 30/80. Mean DLQI value at T0 was 16.4. Patients N. 2 and N. 3 interrupted their previous treatment with biological drugs due to secondary therapeutic ineffectiveness. Patient N. 5 interrupted the treatment with dimethyl fumarate due to the onset of daytime flushing and diarrhea. Patients N. 1 and N. 7 interrupted the cyclosporine due to an increase in mean blood pressure. In all other cases, the previous treatments were suspended due to primary or secondary therapeutic ineffectiveness. All patients were affected by oligoarticular PsA, with a prevalent involvement of the distal interphalangeal joints. The mean patient PGA value at baseline of treatment with tildrakizumab was 6.5; Mean DAPSA value at T0 was 20.75.

Although just patient N. 1 and patient N. 7 had a diagnosis of metabolic syndrome, each patient had at least one metabolic comorbidity treated with multiple drugs (Table 1). One patient also had active bowel diverticulitis undergoing treatment with antibiotics, whilst another patient had been diagnosed with benign prostatic hyperplasia and had been treated with dutasteride. The distribution of comorbidities is reported in Figure 1.

Table 2 shows patient's clinimetric score after 28 weeks of treatment with tildrakizumab 100 mg. PASI at T28 was

Table 1. Characteristics of Enrolled Patients.

PATIENTS	Comorbidities/therapies	Previous treatments for psoriasis/psoriatic arthritis	T0 Pasi	T0 CRP (mg/dl)	T0 PAIN VAS	T0 Hand NAPSI	T0 DLQI	T0 Patient PGA	DAPSA
N. 1 - Male, 52 years old	Hypertension, diabetes, hypercholesterolemia/Olmesartan, metformin, rosuvastatin	Topicals, methotrexate, cyclosporin, methylprednisolone	10	2.31	60	40	14	7	31
N. 2 - Female, 79 years old	Hypertension, hypercholesterolemia/ Bisoprolol + furosemide, lovastatin	Topicals, acitretin, brodalumab	13	1.2	50	0	11	6	19
N. 3 - Male, 63 years old	Hypercholesterolemia/Rosuvastatin + ezetimibe	Topicals, certolizumab pegol	15	0.26	50	40	10	5	14
N. 4 - Male, 49 years old	Hypertension, colon diverticulitis/ Amlodipine/rifaximine	Topicals	20	3.8	40	33	18	9	17
N. 5 - Male, 53 years old	Hypercholesterolemia/ lovastatin	Dimethyl fumarate	10	<0.40	60	20	10	6	17
N. 6 - Female, 54 years old	Hypertension, mixed connective tissue disease/ ramipril + amlodipine	Topicals, cyclosporine	18	1.8	70	0	23	8	32
N. 7 - Female, 62 years old	Hypertension, diabetes, obesity/ metformin, enalapril, nicotinamide	Topicals, dimethyl fumarate, cyclosporine	7	2	30	20	30	4	14
N. 8 - Male, 38 years old	Hypercholesterolemia, obesity, benign prostatic hypertrophy/ Atorvastatin, dutasteride	Topicals	20	4.2	50	0	15	7	22

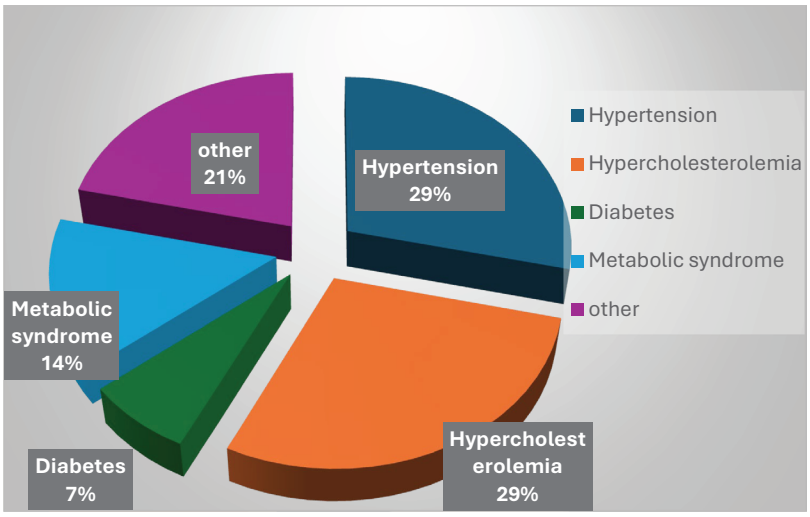

Figure 1. Distribution of comorbidit

Table 2. Evaluation of Clinimetric Scores at Baseline and after 28 Weeks of Treatment.

PATIENT	T28 Pasi	T28 CRP (mg/dl)	T28 PAIN VAS	T28 Hand NAPSI	T28 DLQI	T28 patient PGA	T28 DAPSA
1	0	0.6	0	8	0	0	2
2	3	0.9	0	0	3	2	2
3	3	0.75	0	4	4	1	1
4	12	0.51	0	10	10	7	1
5	3	<0.40	20	0	0	2	9
6	0	1	0	0	0	0	2
7	0	<0.40	0	4	1	1	1
8	0	3.1	10	0	0	1	12

about 2.63, reduced by 81.3% from baseline (P<0.001; Figure 2A). C-reactive protein at T28 remained above the normal limit in only one patient, showing a statistically significant mean value reduction of 66% in all patients (P<0.01; Figure 2B). After 28 weeks only two patients reported still having residual arthritic symptoms, though reduced in severity compared to baseline, with a mean PAIN VAS among all patients of 3.75; the PAIN VAS mean reduction was 85% (P<0.002; Figure 2C). Four patients had residual nail involvement, with a mean NAPSI of 6.5/80, showing a statistically significant reduction (P<0.001; Figure 2D) in the mean value from T0 of ≃ 78%. The DLQI at T28 was 2.25, with a mean reduction of 86% compared to T0 (P<0.001; Figure 2E). The mean patient PGA at T28 was 1.75, with a statistically significant reduction of 73% (P<0.001; Figure 2F).

Mean DAPSA index value at T28 was 3.75, and we also highlight a statistically significant reduction of 82% index between T0 and T28 (t-test P<0.000023; Figure 3).

The monitoring of metabolic parameters did not reveal any abnormality during study protocol in treated patients.

Discussion

The role of IL-23 is crucial to maintaining the Th17 population from Th-1 naïve cells being a central effector involved in the pathogenesis also of PsA. Therefore, it is possible to attribute a primary role to dermal dendritic cells and IL-23 producing keratinocytes, different in terms of "cytokine hierarchy" compared to that of Th17. As the data currently available show the proven efficacy of other IL-23 inhibitors other than tildrakizumab in reducing the signs, symptoms, and progression over time of PsA, they have been approved by both the EMA and the FDA for this indication. Despite the current lack of a valid pharmacodynamic rationale and/or any evidence of ineffectiveness, clinicians cannot yet prescribe tildrakizumab as an on-label treatment for PsA, unlike with other IL-23 inhibitors. The documented efficacy of tildrakizumab on different PsA domains could allow clinicians to promptly intervene on patients suffering from severe psoriasis and metabolic comorbidities by acting early on co-occurring PsA symptoms. In fact, the opportunity of early PsA treatment

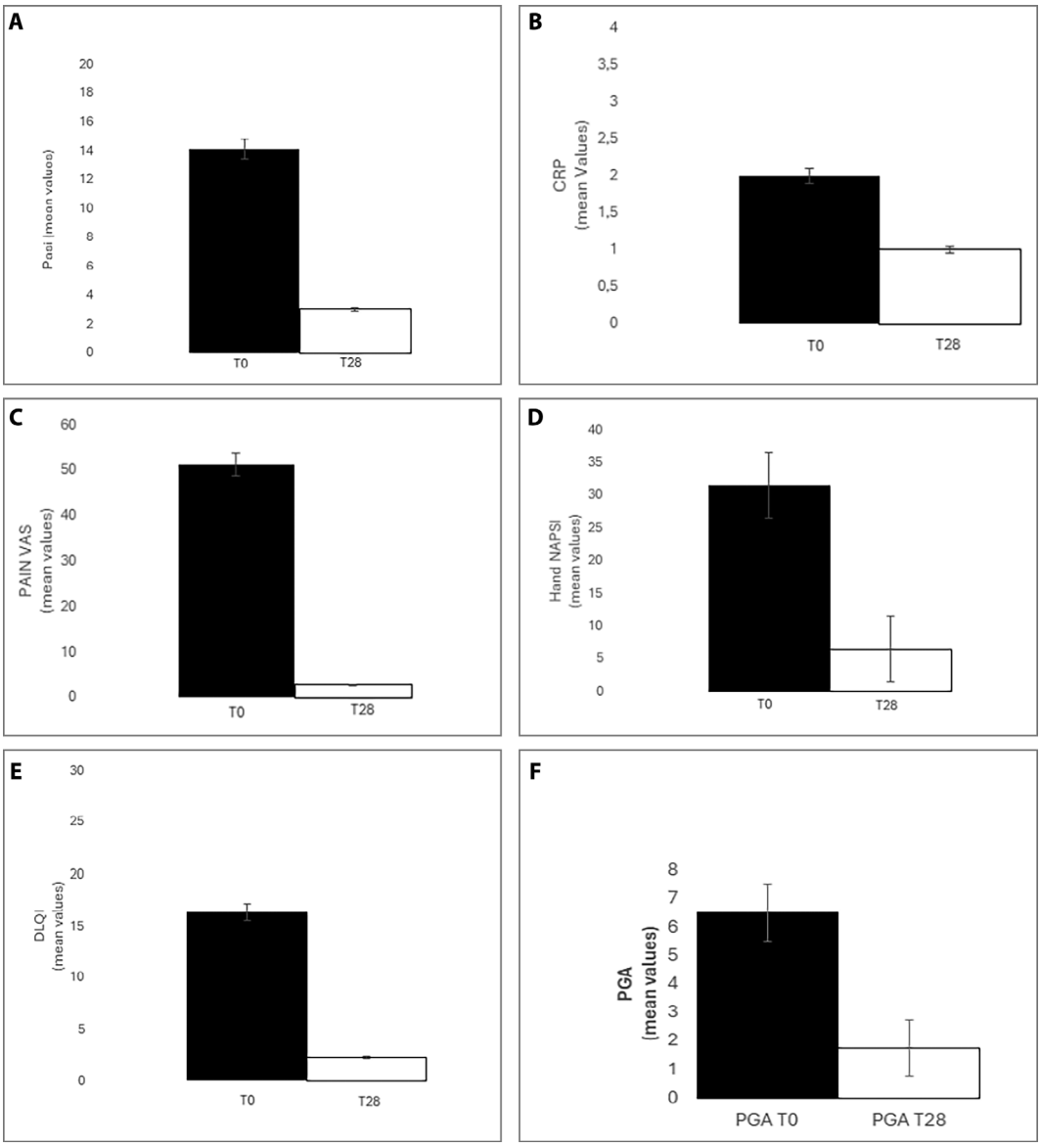

Figure 2. A. Mean PASI values at T0 and T28 (14.13 and 2.63, respectively). Mean reduction: 81.3% (pP<0.001); B. C-reactive protein mean levels at T0 and at T28 (1.95 mg/dl and 0.86 mg/dl, respectively, pP<0.1). We assigned a value of "0" when CRP value was under the lower cut-off limit.; C. Mean PAIN VAS values at T0 and T28 (50 and 3.75, respectively, Pp <0.002); D. Mean HAND NAPSI values at T0 and T28 (30 and 6.5, respectively). Reduction in the mean value from T0 of ≃ 78%, pP<0.001.; E. Mean DLQI values at T0 and T28 (16.4 and 2.25, respectively), mean reduction of 86% compared to T0 (pP<0.001).; F. Mean PGA values at T0 and T28 (6.5 and 1.75, respectively) t-test, pP<0.001.

is now clear, as it can reduce its progression and joint damage, as has been documented in real life studies even in bio-naïve patients [21,30].

Tildrakizumab appears to be safe in patients with cardiovascular comorbidities, including metabolic syndrome, without affecting metabolic parameters.

Dermatologists must also consider the issue of metabolic cardiovascular risk in PsA patients, as shown by our data as well [21]. In fact, some authors have recognized metabolic comorbidities as a possible pathogenetic link with the onset of PsA and not just as a statistically associated disease [31].

Considering biochemical parameters, C-reactive protein is a valid non-specific marker useful to monitoring the progression of joint disease activity, positive in 33–89% of PsA cases, mainly in the early stages of enthesitis [32] but not altered in some active PsA patients[19]. Even in our case series, 25% of patients with active PsA did not show increased CRP values at T0. However, our results also show that only one patient exhibited elevated CRP levels at T28, which remained lower than those recorded at T0. This patient also reported residual, mild arthritic symptoms at T28.

It is noteworthy that the mean PASI at baseline (T0), slightly above 14, did not indicate severe psoriasis. However, the involvement of special sites such as the hands, feet, nails, and genitals accounted for a relatively high mean DLQI at T0 (16.4). After 28 weeks of treatment, PASI was reduced

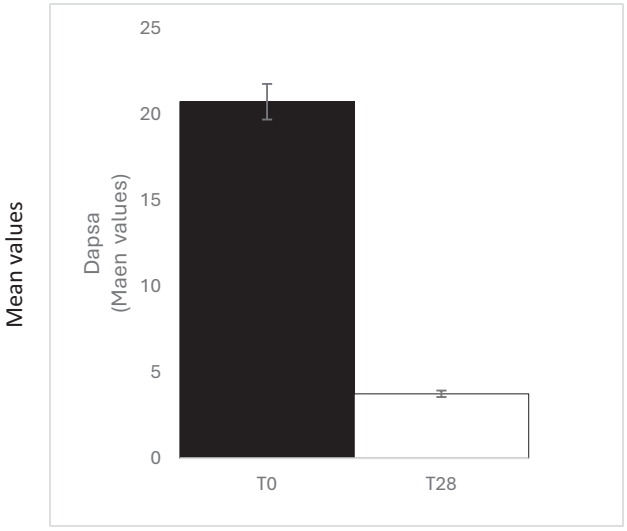

Figure 3. Bar graphs showed statistically significant differences recorded between T0 and T28 for DAPSA score (20.75 and 3.75, respectively). t-test; $P<0.000023$.

by more than 80%, confirming that tildrakizumab can lead to a substantial reduction in disease severity, which was paralleled by a marked improvement in quality of life, as measured by the DLQI.

Nail involvement has been reported in up to 62.5% of psoriatic patients, a finding confirmed in our cohort [16]. The observed improvement in NAPSI was remarkable (approximately 78%), with one patient achieving complete remission of nail psoriasis after 28 weeks. The pathogenesis of nail psoriasis is more closely associated with psoriatic arthritis than with cutaneous psoriasis, and its clinical improvement may reflect better control of joint involvement [16].

Patient PGA assessed by means of VAS is a reliable tool related to both arthritic and cutaneous disease activity [33]. Thus, we decided to evaluate it in order to more accurately highlight the overall trend of the psoriatic pathology over time in each patient. As reported in the results, tildrakizumab was able to strongly reduce the burden of both joint and skin pathology, as reported by the patients, during the 28 weeks of treatment.

The composite DAPSA score is a good tool to assess overall arthritis disease activity as it reflects multiple aspects of affected patients. Our patients obtained a statistically significant reduction in their DAPSA mean score, thus reflecting a better disease course of PsA. It is important to point out that mean baseline score of DAPSA reflected just a mild or moderate activity of PsA.

Anti-TNF-α or anti-IL-17 antibodies are generally prescribed to patients with axial involvement of psoriatic arthritis. Accordingly, no patient with axial disease was treated with tildrakizumab or included in this study. In our clinical practice, our choice of therapeutic option considers firstly anti-IL-17 or anti-TNF-α drugs in patients with a history of psoriatic arthritis of more than one year's duration. These treatment decisions are based on the existing literature, which indicates superior efficacy of these biologics classes compared to anti-IL-23 agents such as tildrakizumab [34].

Psoriasis and PsA are diseases deeply linked to metabolic comorbidities, all having a substrate of chronic systemic inflammation that may play a self-maintaining role [35]. As of March 2025, a dermatologist in Europe can choose the systemic drug for each psoriasis patient from a pool of 18 different therapies, in addition to biosimilars [36]. Among the approved biological drugs for PsA, the ones directed against TNFα show the weakest safety profile, having demonstrated a lower efficacy in obese patients than in normal-weight patients and an association with weight gain in treated patients [37].

Inhibitors of IL-17, while not associated with weight gain in treated patients, have a weaker response in obese patients than in normal-weight patients and are also associated with an increased risk of candidiasis, exacerbated in patients with diabetes mellitus [37,38].

Anti-IL-23 antibodies do not seem to affect glycolipid metabolism or exhibit a different clinical response in obese patients [37]. Recently, tildrakizumab has become available also in a dosage of 200 mg, which is useful in patients weighing over 100 kg and/or with a high disease burden, maintaining the same safety profile compared to the standard dose of 100 mg [39,40]. In light of the RCT tildrakizumab data [23] and based on our real-life clinical experience [21], we consider tildrakizumab to be an effective therapeutic option for psoriasis patients with metabolic comorbidities and signs and symptoms of early PsA who are candidates for biologics to intercept long-term complications such as bone erosions and joint deformities [32].

Conclusions

Awareness of the efficacy of tildrakizumab in PsA could allow clinicians to promptly intervene in patients suffering from severe skin psoriasis and metabolic comorbidities by acting early on co-occurring arthritic symptoms [30].

Further real-life studies are warranted to confirm the role of tildrakizumab in PsA, but our results could be considered a clue of the biological likelihood linked to the treatment of PsA with an IL-23 inhibitor. Our patients achieved complete remission, or at least a dramatic improvement in PsA symptoms after 28 weeks of treatment, together with an almost complete resolution of the skin and nail lesions confirmed by DLQI.

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

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