Journal of Dermatological Treatment

ISSN: 0954-6634 (Print) 1471-1753 (Online) Journal homepage: www.tandfonline.com/journals/ijdt20

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To cite this article: Caterina Lanna, Antonia Rivieccio, Chiara Cattani, Fabio Artosi, Luca Bianchi & Elena Campione (2025) Tildrakizumab 200 mg: a step forward in psoriasis treatment with added metabolic benefits, Journal of Dermatological Treatment, 36:1, 2512159, DOI: 10.1080/09546634.2025.2512159

To link to this article: https://doi.org/10.1080/09546634.2025.2512159

Published online: 15 Jun 2025.

JOURNAL OF DERMATOLOGICAL TREATMENT
2025, VOL. 36, NO. 1, 2512159
https://doi.org/10.1080/09546634.2025.2512159

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RESEARCH ARTICLE

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Tildrakizumab 200 mg: a step forward in psoriasis treatment with added metabolic benefits

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ABSTRACT

Background: Psoriasis is frequently associated with metabolic syndrome and an increased cardiovascular risk. Tildrakizumab, an IL-23 inhibitor, may affect metabolic parameters in addition to improving skin severity.

Aim of the study: To evaluate the impact of increasing tildrakizumab dosage on lipid and glucose levels in psoriasis patients with metabolic syndrome who showed a partial response to the standard 100 mg dose.

Materials and methods: Twenty-five patients with psoriasis and metabolic syndrome were enrolled in a 52-week prospective study. After 16 weeks of treatment with 100 mg tildrakizumab, patients with an absolute PASI >2 were switched to 200 mg. Total cholesterol, LDL, and glucose were measured at baseline, week 16, week 40, alongside PASI and DLQI.

Results: At baseline, mean total cholesterol, LDL, and glucose were 190.7, 120.1, and 99.4 mg/dL, respectively. The 100 mg dose did not result in significant metabolic changes at week 16. However, switching to 200 mg tildrakizumab led to significant reductions at week 40 in total cholesterol (178.3 mg/dL), LDL (110.1 mg/dL), and glucose (87.2 mg/dL) (all $p < 0.05$). Significant improvements in PASI (1.2) and DLQI (0.2) were also observed ($p < 0.05$).

Conclusions: Increasing the tildrakizumab dose to 200 mg in partial responders with metabolic syndrome significantly improved both skin severity and metabolic profiles, lowering cholesterol, LDL, and glucose. These findings suggest a possible dose-dependent effect of tildrakizumab on metabolic parameters through enhanced IL-23 inhibition.

ARTICLE HISTORY
Received 29 April 2025
Accepted 21 May 2025

KEYWORDS
Psoriasis; tildrakizumab; metabolic syndrome

Introduction

Psoriasis is a chronic inflammatory disease affecting approximately 2–3% of the global population, most commonly manifesting as chronic plaque psoriasis (1). This dermatological condition is characterized by the appearance of red, scaly skin patches, often accompanied by pruritus (1). However, psoriasis is not solely a skin disease; it exhibits a systemic nature, also affecting the nails, joints, and other organs, and is associated with significant comorbidities, particularly cardiovascular and metabolic disorders (1). The presence of these comorbidities in a considerable percentage of patients underscores the systemic inflammatory nature of psoriasis. Of particular relevance is the comorbidity between psoriasis and metabolic syndrome, a cluster of interconnected conditions including abdominal obesity, atherogenic dyslipidemia, hypertension, insulin resistance, and hyperglycemia (1). The prevalence of metabolic syndrome in patients with psoriasis is significantly higher than in the general population, with studies reporting rates varying from 16.67% to 39.3% across different populations (1). This high co-occurrence not only increases the overall disease burden but also elevates the risk of adverse cardiovascular events. Therefore, psoriasis is recognized as a systemic inflammatory condition that predisposes individuals to the development of metabolic syndrome (1).

Given the frequent coexistence of psoriasis and metabolic syndrome, it is essential to understand how psoriasis treatments affect the metabolic profile of patients. The presence of both conditions can influence therapeutic recommendations, and systemic treatments for psoriasis, including biological drugs, have the potential to impact metabolic parameters by reducing inflammation and oxidative stress (2–4). It has been observed that some therapies, such as etanercept and adalimumab, can improve metabolic parameters (5). Consequently, evaluating the metabolic impact of psoriasis treatments, especially in patients with preexisting metabolic syndrome, is crucial for optimizing therapeutic decisions and considering both dermatological and metabolic outcomes (2).

Tildrakizumab is a high-affinity humanized IgG1κ monoclonal antibody, approved by the FDA in March 2018 for the treatment of adults with moderate to severe chronic plaque psoriasis who are candidates for systemic therapy or phototherapy (6). Its mechanism of action involves selectively binding to the p19 subunit of interleukin 23 (IL-23), inhibiting its interaction with the IL-23 receptor (6). IL-23 is a key cytokine in the pathogenesis of psoriasis. This inhibition blocks the release of pro-inflammatory cytokines and chemokines, such as IL-17 and TNF-α, which mediate the epidermal hyperplasia, immune activation of keratinocytes, and tissue inflammation typical of psoriasis. Importantly, tildrakizumab does not alter the effects of IL-12, which is crucial for protection against

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infections and cancer (7). The selective targeting of IL-23, a central cytokine in the pathogenesis of psoriasis and potentially linked to metabolic inflammation, makes tildrakizumab a promising therapeutic agent to study for its impact on the metabolic profile in patients with both psoriasis and metabolic syndrome.

The objective of the present study is to evaluate and compare the effect of switching to a higher dose of tildrakizumab (200 mg) compared to maintaining the standard dose (100 mg) on glucose and cholesterol levels in patients with chronic plaque psoriasis and metabolic syndrome who had not achieved an adequate response to the 100 mg dose.

Materials and methods

We conducted a 52-week study at the University Hospital of Rome Tor Vergata involving a total of 25 patients with chronic plaque psoriasis and metabolic syndrome. All patients had concomitant metabolic syndrome and either a body weight above 90 kg or a high disease burden, featuring a Psoriasis Area and Severity Index (PASI) ≥16 or involvement of difficult-to-treat areas (scalp, palms/soles, nails, and genitalia). This group was compared to patients previously treated with tildrakizumab 100 mg for at least 6 months, prior to the reimbursement of tildrakizumab 200 mg in Italy.

Patient eligibility for tildrakizumab treatment was assessed according to the Italian adaptation of the EuroGuiDerm guideline on the systemic treatment of chronic plaque psoriasis. All patients were followed for at least 40 weeks and were classified as either non-responders or partial responders to tildrakizumab 100 mg therapy.

The primary objective was to evaluate the reduction in blood glucose and cholesterol levels with tildrakizumab 200 mg, compared to the same values when patients were treated with tildrakizumab 100 mg. To this aim, blood glucose and cholesterol levels were measured every 12 weeks throughout the study period.

All patients received tildrakizumab 100 or 200 mg according to the Summary of Product Characteristics. The effectiveness of tildrakizumab 100 or 200 mg was evaluated at the 24-week visit in terms of absolute PASI ≤ 2. We also evaluated the decrease in DLQI (Dermatology Life Quality Index). Additionally, the occurrence of any adverse event (AE) was recorded at week 16.

Statistical differences between the values at baseline (Week 0), at Week 16 (during 100 mg treatment), and at Week 40 (after switching to 200 mg) for continuous variables (total cholesterol, LDL cholesterol, glucose, PASI, DLQI) were assessed using the Student's t-test, as appropriate for comparisons between two group means. A *p*-value of 0.05 or less was considered statistically significant. For categorical variables, absolute numbers and percentages were reported. No adjustments were made for multiple comparisons.

The timeline of the study is summarized in Figure 1.

Results

A total of 25 patients, 10 females and 15 males, with an average age of 53.5 years was enrolled. 9 patients were also affected by blood hypertension, 5 patients had diabetes, 10 patients had a BMI >30 (obesity grade I), and 8 patients dyslipidemia.

At week 0 the average level of total cholesterol was 190.7 mg/dL (SD 33.4 mg/dL), while LDL was 120.1 mg/dL (SD 31.4 mg/dL), glucose was 99.4 mg/dL (SD 10.4 mg/dL). After 16 weeks of tildrakizumab 100 mg administration there weren't any statistical significative differences in term of cholesterol, LDL, or glucose reduction.

After 24 weeks of tildrakizumab 100 mg there wasn't any statistical significative differences in term of cholesterol, LDL and glucose (*p*-value > 0.05).

All the patients were therefore switched to tildrakizumab 200 mg. After 16 weeks of tildrakizumab 200 mg (total weeks: 40), the average level of total cholesterol was 178.3 mg/dL (SD 32 mg/dL), LDL was 110.1 mg/dL (SD 27 mg/dL), glucose was 87.2 mg/dL (SD 12 mg/dL). There was a significant reduction in these parameters comparing between Week 0 (Week24 considering the whole study) of tildrakizumab 200 mg and between week 0 tildrakizumab 100 mg ($p < 0.05$) (Figures 2–4).

Regarding PASI evaluation, the average PASI at tildrakizumab 100 mg week 0 was 15.4 and after 24 weeks 8.15, after the switch to tildrakizumab 200 mg, at week 40 the average PASI was 1.2 (Figure 5).

Regarding DLQI evaluation, the average DLQI at tildrakizumab 100 mg week 0 was 10.6 and after 24 weeks 6.8, after the switch to tildrakizumab 200 mg, at week 40 the average DLQI was 0.2 (Figure 6).

No adverse events were observed at week 16 and during all the study.

Discussion

The main results of this study indicate that patients with chronic plaque psoriasis and metabolic syndrome who had not responded

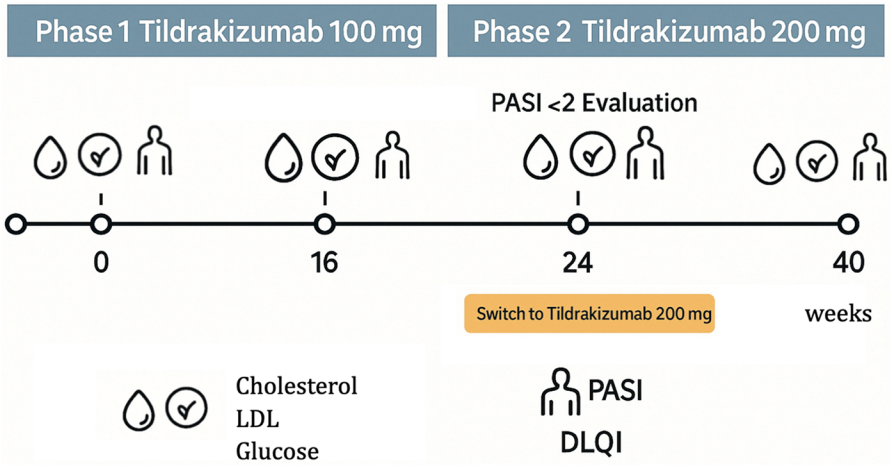

Figure 1. Study timeline.

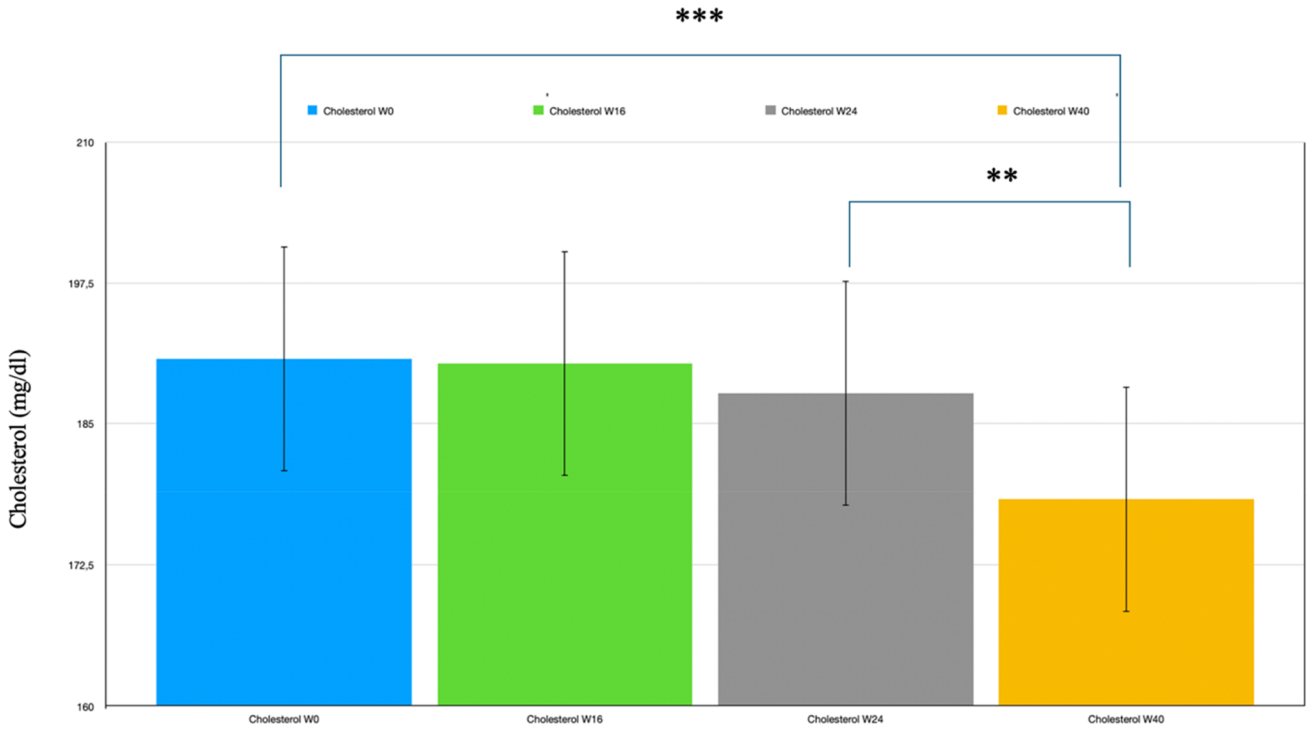

Figure 2. Trend of total cholesterol levels (mg/dL) at Week 0, Week 16, Week 24, and Week 40. Bars represent mean values with standard deviation. **p-value <0.01, ***p-value <0.001. Legend: Blue bar: Cholesterol Week 0; green bar: Cholesterol Week 16; gray bar: Cholesterol Week 24; yellow bar: Cholesterol Week 40.

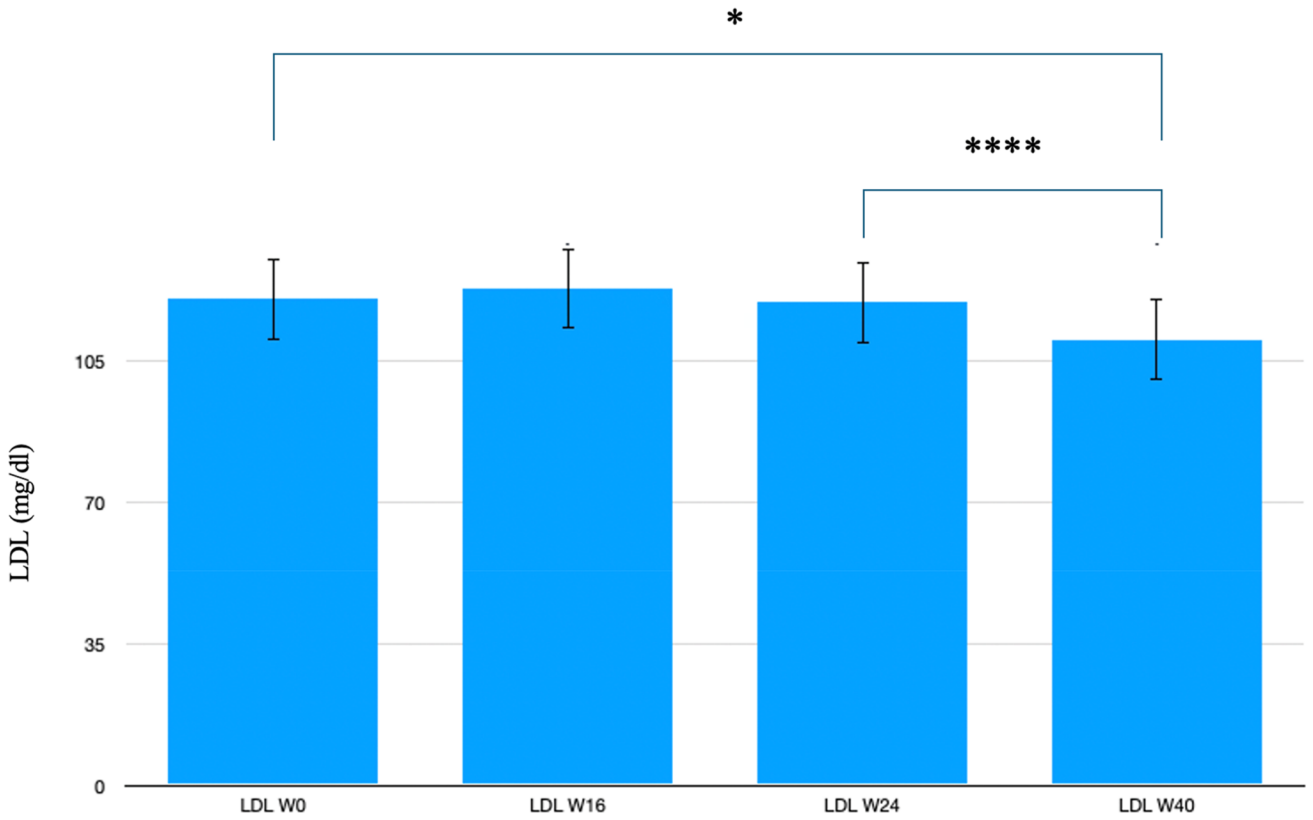

Figure 3. Trend of LDL cholesterol levels (mg/dL) at Week 0, Week 16, Week 24, and Week 40. Bars represent mean values with standard deviation. *p-values <0.05, ****p-value <0.0001. Legend: Blue bar: LDL Week 0; light blue bar: LDL Week 16; gray bar: LDL Week 24; dark blue bar: LDL Week 40.

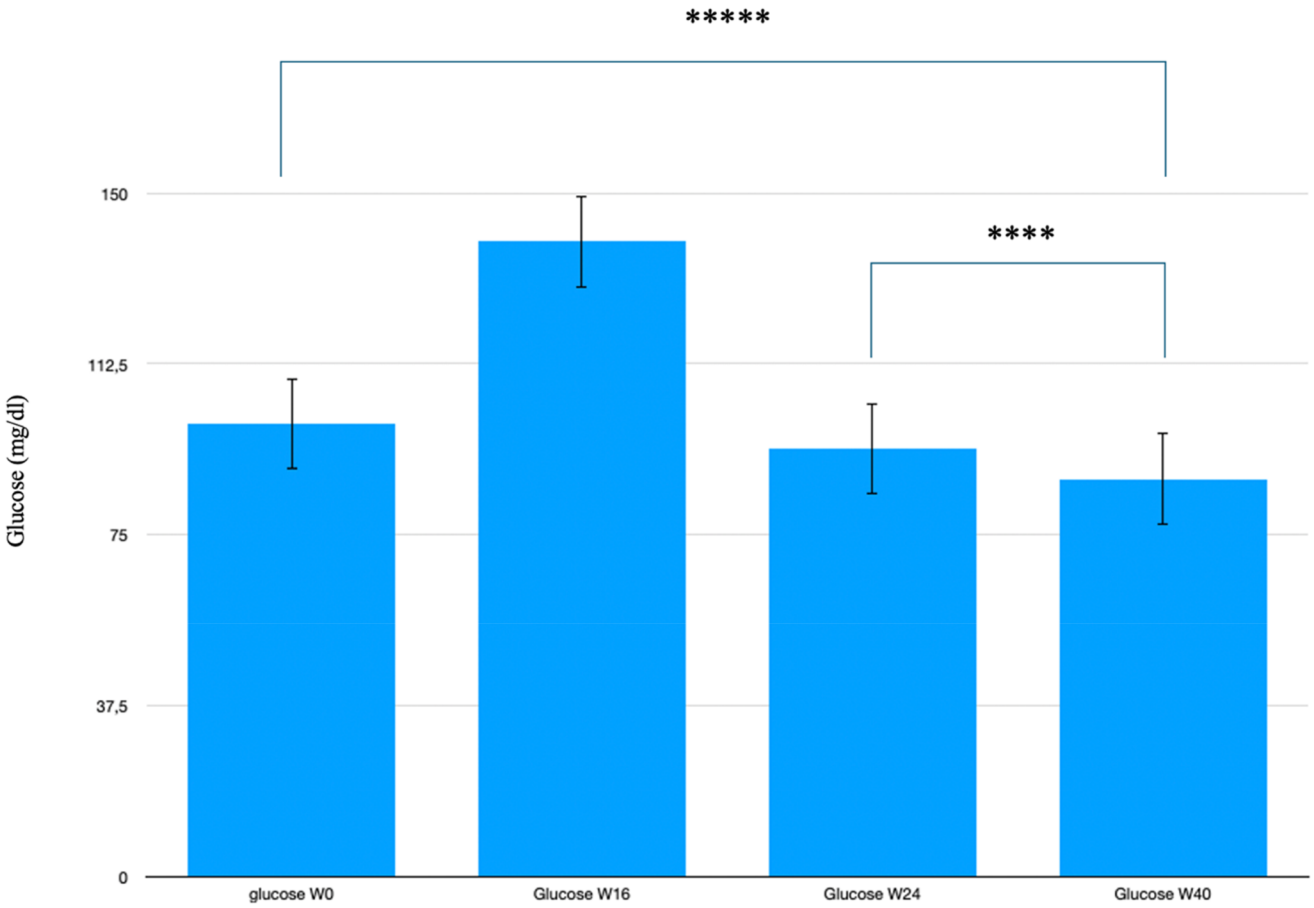

Figure 4. Trend of glucose levels (mg/dL) at Week 0, Week 16, Week 24, and Week 40. Bars represent mean values with standard deviation. ****p-value <0.0001, *****p-value <0.00001. Legend: Blue bar: Glucose Week 0; light blue bar: Glucose Week 16; gray bar: Glucose Week 24; dark blue bar: Glucose Week 40.

or had only partially responded to tildrakizumab 100 mg experienced a statistically significant reduction in total cholesterol, LDL cholesterol, and fasting glucose levels after switching to tildrakizumab 200 mg. This suggests that a greater inhibition of IL-23 may have a more pronounced effect on metabolic parameters in this specific patient population.

The existing literature on the impact of tildrakizumab on metabolic parameters in patients with psoriasis, with or without metabolic syndrome, presents conflicting results. A post-hoc analysis of 5-year data from the reSURFACE 1 and reSURFACE 2 studies demonstrated that tildrakizumab (100 mg and 200 mg) maintains efficacy and a favorable safety profile in patients with psoriasis, regardless of the presence of metabolic syndrome (8). However, this analysis did not specifically focus on changes in glucose and cholesterol levels as primary endpoints. Another post-hoc analysis of the same studies examined the effect of tildrakizumab on cardiometabolic risk factors in patients with moderate to severe psoriasis with and without metabolic syndrome (9). This study found limited changes in these risk factors following treatment with tildrakizumab, with variations generally similar regardless of dose or metabolic syndrome status. Although mean changes in fasting glucose were numerically decreased in patients with metabolic syndrome receiving 100 mg, overall changes in glucose and other lipid parameters were not statistically significant or consistently dose-dependent (9).

Despite previous research suggesting that tildrakizumab at standard doses may not significantly alter cardiometabolic risk factors in all patients with psoriasis, the results of the present study, showing significant reductions in cholesterol and glucose with a higher dose in non-responder patients with metabolic syndrome, indicate a potential differential effect in this specific subgroup. This could be attributed to the fact that the higher dose achieves a greater degree of IL-23 inhibition, which may be necessary to elicit metabolic benefits in these treatment-resistant patients. Indeed, it has been already reported that tildrakizumab 200 mg has more efficacy in patients with a body weight ≥ 90 kg or high disease burden (10).

Comparing the effects of other biological drugs (anti-TNF-α, anti-IL-17, anti-IL-12/23) on the metabolic profile in patients with psoriasis and metabolic syndrome reveals variability depending on the specific drug and the patient population (5). Etanercept and adalimumab (TNF-α inhibitors) have been shown to improve metabolic parameters, including blood lipid and glucose levels (5). One study comparing anti-IL-17 and anti-IL-23 biologics with cyclosporine demonstrated that both classes of biologics improved blood glucose and lipid profiles (11). In particular, it was suggested that anti-IL-23 biologics may have better long-term efficacy in patients with metabolic syndrome (11). Another research report indicated that various biologics (including IL-17 and IL-23 inhibitors) improved HDL, CRP, and ESR levels and reduced the number of patients meeting the criteria for metabolic syndrome (12). However, one study found no significant changes in biochemical metabolic parameters with anti-IL-12/23 or anti-IL-17 therapies over one year (13).

The potential mechanisms through which tildrakizumab 200 mg might have influenced the metabolic profile in these patients lie in

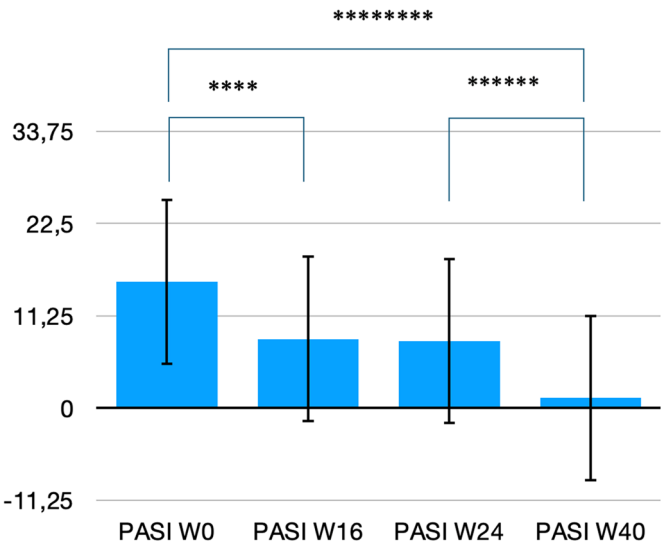

Figure 5. Trend of PASI score at Week 0, Week 24 (with 100 mg), and Week 40 (after switching to 200 mg). Bars represent mean values. ****p-value < 0.0001, ******p-value < 0.000001, ********p-value <0.00000001.

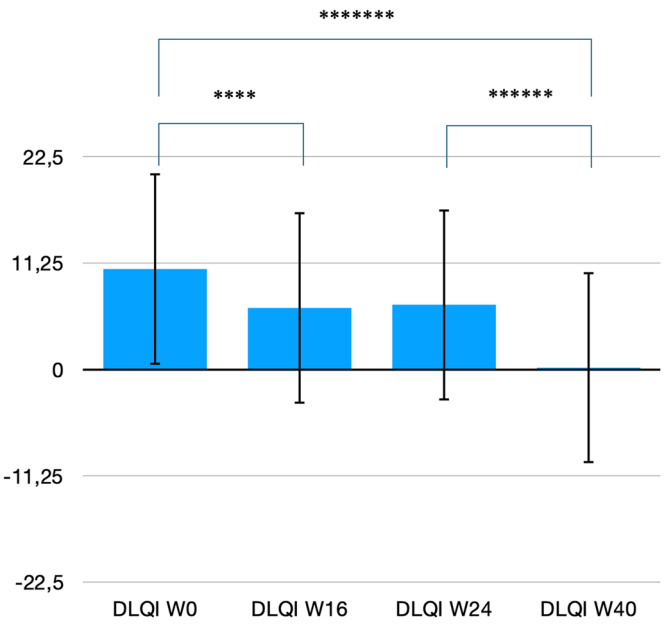

Figure 6. Trend of DLQI score at Week 0, Week 24 (with 100 mg), and Week 40 (after switching to 200 mg). Bars represent mean values. ****p-value <0.0001, ******p-value <0.000001, *******p-value 0.0000001.

the well-established link between the IL-23/Th-17 pathway and both psoriasis and metabolic syndrome (14–16). Dysregulation of this pathway is central to both pathologies and may promote susceptibility to metabolic and cardiovascular diseases (14). Tildrakizumab, by inhibiting IL-23, could disrupt these signals, potentially reducing both the psoriatic inflammatory burden and the risk of developing atherosclerosis and cardiometabolic diseases (14).

The reduction in cholesterol and glucose levels with the higher dose of tildrakizumab could be mediated by a more effective suppression of the IL-23/Th-17 inflammatory axis. This could lead to a decrease in systemic inflammation, which is known to contribute to insulin resistance and dyslipidemia The higher dose might be necessary to overcome the inflammatory environment in patients who did not fully respond to the standard dose (17). The observed decrease in LDL cholesterol could be linked to findings suggesting a positive association between IL-23 expression in adipose tissue and LDL cholesterol levels, and an inverse association with adiponectin. A more effective blockade of IL-23 with the higher dose could lead to a reduction in LDL-c and potentially an increase in adiponectin. However, findings that IL-23 deficiency in mice worsened metabolic parameters suggest a complex role for IL-23, and the higher dose of tildrakizumab might be needed to shift the balance in patients with psoriasis and metabolic syndrome (18).

The improvement in PASI and DLQI scores observed with the higher dose of tildrakizumab likely reflects a significant reduction in the inflammatory burden of psoriasis. This reduction in systemic inflammation could be a key factor in the concurrent improvement of the metabolic profile, suggesting a strong interaction between skin inflammation and metabolic health in these patients (19). Patients with metabolic syndrome often present with more severe psoriasis and a reduced response to treatment (2). The improvement observed with the higher dose suggests that better control of psoriasis in this subgroup could have a positive effect on their metabolic health.

Recent findings by Kochumon et al. demonstrate increased IL-23 expression in subcutaneous adipose tissue of individuals with elevated LDL cholesterol, accompanied by upregulation of TNF-α, IL-12, IL-18, and associated chemokines. These data suggest that IL-23 contributes to local and systemic inflammation *via* recruitment of immune cells and amplification of the Th17/IL-17 axis, potentially impairing insulin signaling, leading to reduced insulin sensitivity in peripheral tissues (17). IL-23 also induces β cell loss, through inflammatory cell infiltration, increased apoptosis, and altered cytokine expression in the pancreatic islets. These findings support a contributory role for IL-23 in the pathogenesis of diabetes (20).

IL-23 is increasingly recognized as a critical mediator in the pathogenesis of vascular diseases. Elevated serum levels of IL-23 are associated with atherosclerotic changes in the arteries of the lower extremities. Mechanistically, IL-23 contributes to endothelial dysfunction by enhancing the recruitment and adhesion of immune cells to vessel walls, thereby facilitating the inflammatory processes that underlie atherosclerosis (21).

Given the established role of IL-23 in psoriasis pathogenesis and the strong epidemiological link between psoriasis and metabolic syndrome, these findings support IL-23 as a potential common denominator in cutaneous and metabolic inflammation. Targeting IL-23 may therefore offer therapeutic benefit beyond the skin, addressing systemic inflammatory components in psoriatic disease.

Several limitations should be considered when interpreting the findings of this study. Primarily, the retrospective and uncontrolled nature of the design means that while we observed significant metabolic improvements following the dose escalation to 200 mg of tildrakizumab, we cannot definitively establish a cause-and-effect relationship. It is possible that other factors, such as concurrent lifestyle changes or modifications in other medications the patients were receiving for their metabolic syndrome, may have contributed to the observed benefits. Additionally, the relatively small sample size ($n = 25$) limits the statistical power of the study and the generalizability of our results to a broader population of patients (22).

Future research should include larger, multicenter, randomized, and controlled studies to validate these preliminary results and provide more robust evidence on the effect of tildrakizumab dosage on metabolic profiles in patients with psoriasis and metabolic

syndrome. Further investigation into the specific mechanisms through which greater IL-23 inhibition could lead to improvements in glucose and cholesterol metabolism in this patient population is also recommended, potentially through detailed analysis of inflammatory markers, adipokines, and insulin sensitivity. It would also be useful to explore whether these metabolic improvements translate into better long-term cardiovascular outcomes in this high-risk group.

Conclusion

In conclusion, the results of the present study suggest that switching to tildrakizumab 200 mg can lead to a significant reduction in total cholesterol, LDL cholesterol, and fasting glucose levels in patients with chronic plaque psoriasis and metabolic syndrome who had not responded adequately to the 100 mg dose. These findings, while preliminary due to the study's limitations, highlight the potential of more intensive IL-23 inhibition to improve not only dermatological outcomes but also the metabolic profile in this high-risk patient population. Further research, through larger and controlled studies, is warranted to confirm these results and elucidate the underlying mechanisms, potentially paving the way for new therapeutic strategies for the integrated management of psoriasis and its metabolic comorbidities.

Ethics statement

This retrospective study based on routine clinical practice data did not require ethics committee approval.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Funding

This study was funded by Almirall.

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