Open Journal of
Clinical & Medical
Case Reports

Volume 12 (2026)
Issue 5

Case Report ISSN: 2379-1039

Synergistic dual biologic therapy targeting il-23 and il-31 pathways in a patient with coexisting psoriasis and prurigo nodularis

Fatima Escalera, BA*; Danilo C Del Campo, MD, FAAD

***Corresponding Author: Fatima Escalera**
Chicago Skin Clinic, USA.
Email: fescalera13@gmail.com

Abstract

Prurigo nodularis and psoriasis are distinct immune-mediated skin diseases with overlapping clinical features but divergent cytokine drivers, namely IL-31 and IL-23, respectively, posing diagnostic and therapeutic challenges when they coexist. A 52-year-old Hispanic male with a four-year history of well-controlled plaque psoriasis, managed with risankizumab, presented with new-onset Prurigo Nodularis (PN). Given the emergence of PN despite stable psoriatic disease, dual biologic therapy was initiated: the patient continued his IL-23 inhibitor (risankizumab) while starting nemolizumab, an IL-31 receptor antagonist. Within four weeks of starting nemolizumab, the patient experienced significant symptomatic relief, marked improvement in nodular lesions and pruritus, and resolution of postinflammatory hyperpigmentation. This case report is the first, to our knowledge, to document the successful dual biologic treatment of coexisting psoriasis and prurigo nodularis, highlighting a rare phenomenon of a superficial dermatologic shift where a patient with confirmed psoriasis later developed PN. It supports a strategic shift toward complementary immunologic targeting—specifically IL-23 and IL-31 inhibition—in managing overlapping chronic inflammatory dermatoses, moving beyond the traditional single-disease focus.

Introduction

Prurigo Nodularis (PN) is a chronic pruritic skin disorder characterized by intensely itchy, hyperpigmented nodules that develop often as a consequence of repeated scratching [1,2]. The pathogenesis of PN involves complex neuroimmune dysregulation, including elevated levels of Interleukin-31 (IL-31), a key mediator of pruritus, and interactions between the peripheral nervous system and the immune system, contributing to the persistent itch–scratch cycle [1-3]. PN significantly impairs quality of life, leading to sleep disturbances, psychological distress, and social limitations [1,2]. Conventional topical therapies, such as corticosteroids, frequently fail to provide adequate control in moderate-to-severe disease, necessitating

systemic treatment approaches [4]. Recent randomized controlled trials have demonstrated that IL-31 receptor blockade with nemolizumab can substantially reduce pruritus and nodular lesion burden, offering a promising targeted therapy for patients with refractory PN [4-6].

In contrast, psoriasis is a chronic, immune-mediated inflammatory skin disorder characterized by well-demarcated erythematous plaques with overlying silvery scales, affecting up to 3% of the global population [7]. The pathogenesis is driven by IL-23–mediated activation of T Helper 17 (Th17) cells, resulting in keratinocyte hyperproliferation, local inflammation, and systemic immune activation [7,8]. Psoriasis can be managed with topical agents, phototherapy, systemic immunomodulators, and biologic therapies targeting critical inflammatory pathways, including IL-17 and IL-23 [7-9]. Biologics such as risankizumab, an IL-23 inhibitor, have demonstrated robust clinical efficacy and favorable safety profiles in achieving sustained disease control in moderate-to-severe plaque psoriasis [7].

Although PN and psoriasis are clinically and immunologically distinct, both represent immune-driven skin disorders that can profoundly affect patient quality of life. The coexistence of these conditions is uncommon and poses a significant therapeutic challenge, as treatment strategies must simultaneously address distinct inflammatory pathways and debilitating pruritus [10]. Conventional monotherapy may be insufficient, highlighting the potential need for combination or dual biologic therapy.

In this case report, we present a patient with well-controlled plaque psoriasis on risankizumab who subsequently developed prurigo nodularis and was successfully treated with the addition of nemolizumab. Within three months of dual therapy, the patient experienced complete resolution of pruritus and marked improvement in nodular lesions, illustrating the potential efficacy and safety of concurrent IL-23 and IL-31 blockade. This case underscores the emerging role of mechanism-based combination biologic therapy in managing complex, coexisting inflammatory dermatologic conditions and contributes to the limited literature on dual biologic treatment strategies for overlapping skin diseases [11,12].

Case Presentation

A 52-year-old Hispanic male with a long-standing history of plaque psoriasis had been well maintained on risankizumab for the past four years, with stable disease control and no recent flares of psoriasis.

During a routine follow-up, the patient reported the recent onset of intensely pruritic, dark nodular lesions. As seen in **Figures 1,3, and 5**, physical examination revealed over 20 hyperpigmented, excoriated nodules distributed symmetrically across the bilateral upper extremities, trunk, and lower extremities. Psoriatic plaques remained stable and noninflamed.

The patient was diagnosed with Prurigo Nodularis (PN) and failed multiple topical corticosteroid regimens. Given the severity of his symptoms and their significant impact on his quality of life, biologic therapy with nemolizumab was initiated. Before treatment, the patient reported significant emotional distress and self-consciousness, mirroring his earlier experiences with psoriasis. Risankizumab had

previously stabilized his psoriasis, but the emergence of PN brought back feelings of distress due to the visible, itchy nodules. Since topicals were ineffective, dual therapy with nemolizumab and risankizumab was pursued.

The patient received a loading dose of two subcutaneous injections administered to the left anterior thigh, followed by self-administered monthly maintenance dosing at home. He continued his existing risankizumab regimen without interruption [6].

At the three-month follow-up, the patient reported complete resolution of pruritus and improved skin texture **(Figures 2,4, and 6)**. Examination showed softening and flattening of PN lesions. The patient noted better sleep and expressed excitement for the summer.

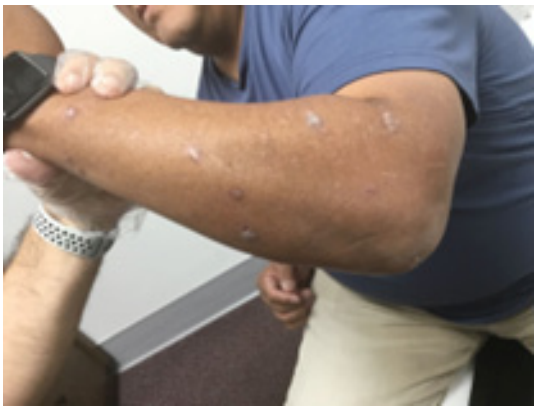

Figure 1: Clinical image of the patient's left arm prior to treatment, demonstrating multiple excoriated and hyperkeratotic nodules consistent with prurigo nodularis. Lesions are distributed along the extensor surface of the forearm and elbow, with evidence of lichenification, post-inflammatory hyperpigmentation, and excoriation. The patient reported persistent pruritus and chronic scratching.

Figure 2: Follow-up image of the same patient's left arm three months after initiating nemolizumab therapy. Marked clinical improvement is observed, with resolution of active prurigo nodularis lesions. The skin appears smooth with minimal post-inflammatory hyperpigmentation and no evidence of excoriation, nodularity, or lichenification. The patient reported complete clearance of lesions and significant reduction in pruritus.

Figure 3: Baseline image of the patient's occipital scalp and upper posterior neck prior to treatment, demonstrating an erythematous and hyperkeratotic nodule consistent with prurigo nodularis. Lesion is firm, excoriated, and located along the hairline. The patient reported intense pruritus and frequent scratching in this area, contributing to lesion persistence and inflammation.

Figure 4: Three-month follow-up image of the occipital scalp and posterior neck following initiation of nemolizumab therapy. The previously present prurigo nodularis lesions have resolved, with no visible nodules or active inflammation. The skin appears smooth with minimal residual post-inflammatory changes. The patient reported full clearance of scalp lesions and complete cessation of pruritus in this area.

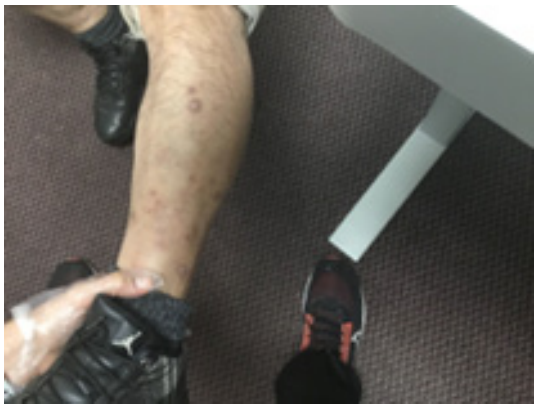

Figure 5: Baseline image of the patient's lower left leg prior to nemolizab treatment, demonstrating hyperkeratotic nodules on the anterior shin, consistent with prurigo nodularis. The lesions are firm, hyperpigmented, and excoriated. The patient confirms the presence of intense pruritus and frequent scratching.

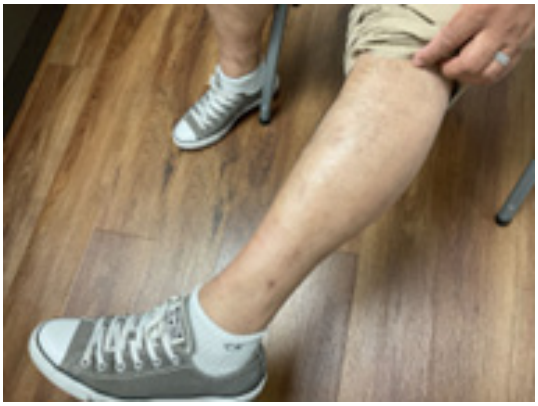

Figure 6: Three-month follow-up image of the patient's anterior lower left leg following initiation of nemolizumab therapy. The previously present prurigo nodularis lesions have resolved, with no visible nodules or active inflammation. The skin appears smooth with minimal residual post-inflammatory changes. The patient reported full clearance of leg lesions and complete cessation of pruritus in this area.

Discussion

This case highlights the evolving landscape of immune-mediated dermatologic conditions and the emerging role of dual biologic therapy [11,12]. Our patient maintained stable plaque psoriasis for over four years with risankizumab, an IL-23 inhibitor, yet developed new-onset prurigo nodularis, raising questions about immune axis shifts and underlying pathophysiology.

The development of prurigo nodularis in a patient with well-controlled psoriasis on risankizumab raises the possibility of an underlying immunologic shift, particularly from Th1/Th17-dominant inflammation toward a Th2-predominant immune response [10-12]. This shift is not fully understood but has been proposed in the context of evolving immune responses, chronic antigen exposure, and selective cytokine inhibition [10].

Psoriasis is traditionally considered a Th1 and Th17-mediated disease, characterized by elevated levels of interferon-γ, IL-17, and IL-23, which drive keratinocyte hyperproliferation and chronic inflammation [7]. Biologic therapies like risankizumab, which selectively inhibit the p19 subunit of IL-23, are highly effective in disrupting this pathway and achieving clinical remission [7].

In contrast, PN is increasingly recognized as a Th2-skewed disorder, with central roles for IL-4, IL-13, and IL-31 [1-4]. IL-31 in particular is directly linked to chronic pruritus, neural remodeling, and epidermal barrier dysfunction [1-6]. Histopathologically, PN is distinguished by skin fibrosis, vertically aligned collagen in the papillary dermis, and neural hyperplasia, features which are not typically seen in psoriasis [2-4].

When a single cytokine axis such as IL-23 is selectively inhibited, it is hypothesized that immune compensation may occur, favoring upregulation or dysregulation of alternative helper T-cell subsets [10].

This phenomenon, often referred to as immune deviation or immunologic plasticity, may lead to a relative Th2 dominance, especially in genetically or environmentally predisposed individuals [10].

The patient's transition from a Th1/Th17 phenotype (psoriasis) to a Th2-dominant Phenotype (PN) illustrates this dynamic interplay between immune pathways. It also reinforces the importance of identifying and treating the active cytokine driver in immune-mediated dermatoses, rather than assuming stable immunologic dominance across the disease course [10-12].

The success of nemolizumab in this case, specifically targeting the IL-31 receptor A, demonstrates how targeted biologic intervention along the Th2 axis can be both effective and safe, even in patients concurrently treated with Th1/Th17-axis inhibitors [10-12]. The non-redundant, complementary targeting of IL-23 and IL-31 represents a rational and mechanistically grounded approach in complex or evolving immune presentations.

Conclusions

In this case, dual biologic therapy targeting IL-23 and IL-31 pathways was both effective and well tolerated in a patient with coexisting plaque psoriasis and prurigo nodularis. The combination of risankizumab and nemolizumab provided rapid symptomatic relief, improved skin texture, and maintained psoriasis control without adverse events.

This case highlights the potential of mechanism-based dual biologic therapy as a viable option in managing complex or overlapping immune-mediated dermatologic conditions, particularly in patients who have failed conventional treatments. While further studies are needed to assess long-term safety, efficacy, and cost-effectiveness, this approach may represent a promising direction in personalized dermatologic care.

References

1. Kwatra SG, Ständer S, Yosipovitch G, Kim BS, Levit NA, O'Malley JT. Pathophysiology of Prurigo Nodularis: Neuroimmune Dysregulation and the Role of Type 2 Inflammation. J Invest Dermatol. 2025; 145: 249-256.

2. Marshall JS, Warrington R, Watson W, Kim HL. An introduction to immunology and immunopathology. Allergy Asthma Clin Immunol. 2018; 14: 49.

3. Garcovich S, Maurelli M, Gisondi P, Peris K, Yosipovitch G, Girolomoni G. Pruritus as a distinctive feature of type 2 inflammation. Vaccines. 2021; 9: 303.

4. Müller S, Zeidler C, Ständer S. Chronic Prurigo Including Prurigo Nodularis: New Insights and Treatments. Am J Clin Dermatol. 2024; 25: 15-33.

5. Licata G, Arisi M, Giorgio CM, et al. Emerging Therapies in the Treatment of Prurigo Nodularis: Biological Therapy and Systematic Review of Literature. Dermatol Ther (Heidelb). 2025; 15: 1705-1718.

6. Megna M, D'Agostino M, Feo F, Ventura V, Tommasino N, Potestio L. IL-23 inhibitors in psoriasis: what have we learnt so far? J Inflamm Res. 2026; 19: 540848.

7. Potestio L, Martora F, Lauletta G, Vallone Y, Battista T, Megna M. The Role of Interleukin 23/17 Axis in Psoriasis Management: A Comprehensive Review of Clinical Trials. Clin Cosmet Investig Dermatol. 2024; 17: 829-842.

8. Pang Y, D'Cunha R, Winzenborg I, Veldman G, Pivorunas V, Wallace K. Risankizumab: Mechanism of action, clinical and translational science. Clin Transl Sci. 2024; 17: e13706.

9. Ständer S, Yosipovitch G, Legat FJ, et al. Efficacy and Safety of Nemolizumab in Patients with Moderate to Severe Prurigo Nodularis: The OLYMPIA 1 Randomized Clinical Phase 3 Trial. JAMA Dermatol. 2025; 161: 147-156.

10. Bianco M, D'Oria F, Falcidia C, et al. New and Emerging Biologics and Jak Inhibitors for the Treatment of Prurigo Nodularis: A Narrative Review. Medicina (Kaunas). 2025; 61: 631.

11. Kaszycki MA, Pixley JN, Feldman SR. Concurrent Atopic Dermatitis and Psoriasis Successfully Treated With Dual Biologic Therapy. Cutis. 2023; 112: E13-E16.

12. Ma L, Chen X, Aziz MAA, Chen A, Cai T, Chen S. Dual Biologic Therapy for Psoriasis in a Patient with Atopic Dermatitis. Psoriasis (Auckl). 2025; 15: 159-161.

Manuscript Information: Received: May 11, 2026; Accepted: June 15, 2026; Published: June 30, 2026

Authors Information: Fatima Escalera, BA*; Danilo C Del Campo, MD, FAAD
Chicago Skin Clinic, USA.

Citation: Escalera F, Del Campo DC. Synergistic dual biologic therapy targeting il-23 and il-31 pathways in a patient with coexisting psoriasis and prurigo nodularis. Open J Clin Med Case Rep. 2026; 2423.