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Secukinumab-Induced Bullous Pemphigoid: A Case Report

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Abstract | **Case Report**

Background: Drug-induced bullous pemphigoid is a rare cutaneous adverse reaction linked to various systemic medications. Secukinumab, an interleukin-17A inhibitor widely used for moderate-to-severe psoriasis, has rarely been implicated in the development of bullous pemphigoid. ***Case Presentation:*** A 79-year-old male with a 20-year history of severe plaque psoriasis—previously recalcitrant to topical corticosteroids and methotrexate—was treated with secukinumab. After 10 months of successful therapy, treatment was interrupted for 3 months due to poor adherence before being resumed. One month post-reinitiation, the patient developed intense pruritus and tense bullous lesions on the upper and lower extremities, rapidly generalizing within days. Histopathological examination revealed a subepidermal split, and direct immunofluorescence demonstrated linear, homogeneous deposition of C3 and IgG along the basement membrane zone, confirming bullous pemphigoid. Treatment with topical corticosteroids and oral doxycycline led to significant clinical improvement within two weeks. However, re-challenge via his subsequent scheduled secukinumab injection triggered a rapid flare of bullous lesions. Definitive discontinuation of secukinumab resulted in lesion clearance and complete resolution. ***Conclusion:*** Clinicians should remain vigilant regarding the rare potential of interleukin-17A inhibitors, such as secukinumab, to induce autoimmune blistering conditions like bullous pemphigoid, particularly following drug reintroduction or interruption.

Keywords: learning disabilities; medication review; primary care; antipsychotics; quality improvement; STOMP.

INTRODUCTION

Bullous pemphigoid is an autoimmune subepidermal blistering disease predominantly affecting the elderly population [1]. Drug-induced bullous pemphigoid accounts for a subset of these cases and typically manifests within three months following medication initiation or reintroduction. Frequently implicated causative agents include diuretics such as furosemide and spironolactone, nonsteroidal anti-inflammatory drugs, amoxicillin, gliptins, programmed cell death protein 1 or ligand 1 inhibitors, and tumor necrosis factor-alpha inhibitors. Secukinumab-induced bullous pemphigoid remains an extremely rare clinical entity [2]. Here, we present a distinct case of bullous pemphigoid triggered by secukinumab re-challenge in a patient with plaque psoriasis.

CASE PRESENTATION

A 79-year-old male presented with a 20-year history of plaque psoriasis that had previously failed to respond adequately to topical corticosteroids and systemic methotrexate. He was subsequently initiated on secukinumab, achieving a notable clinical response over a 10-month period. Due to patient non-compliance, treatment was interrupted for three months before being resumed.

One month after restarting secukinumab, while his psoriasis remained well-controlled, the patient developed severe pruritus accompanied by tense bullous lesions on both upper and lower extremities. The eruption generalized rapidly over a few days.

Histopathological evaluation of a bullous skin biopsy confirmed a subepidermal split. Direct immunofluorescence demonstrated linear, homogeneous deposition of C3 and IgG at the basement membrane zone, establishing the diagnosis of bullous pemphigoid.

The patient was managed with high-potency topical corticosteroids and oral doxycycline, achieving significant clinical recovery within two weeks. However, upon receiving his next scheduled secukinumab injection, the patient experienced an acute relapse of bullous lesions. Secukinumab was permanently discontinued, after which the eruption rapidly resolved without further recurrence.

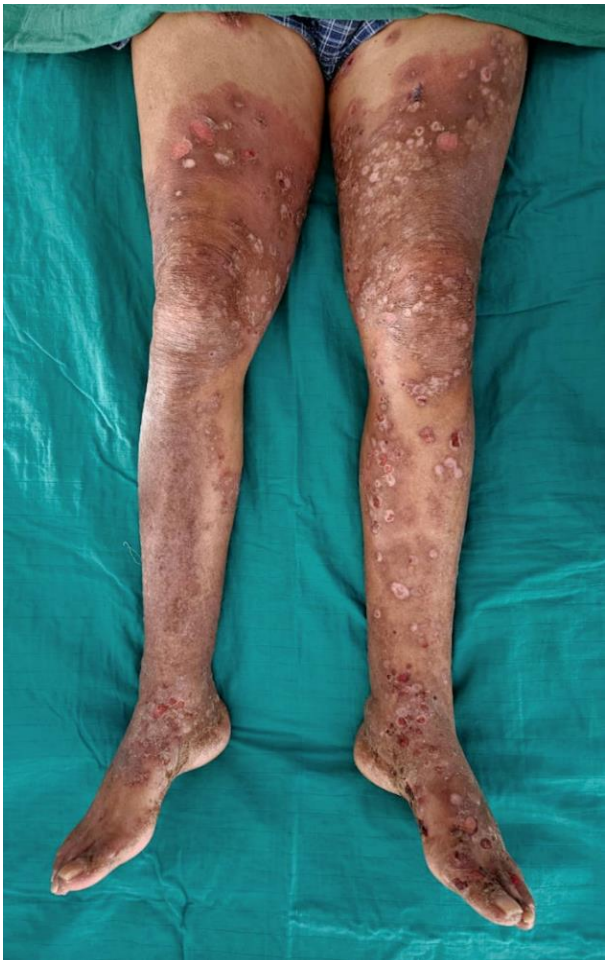

Figure 1: Post bullous erosions on the legs with some psoriasis lesions

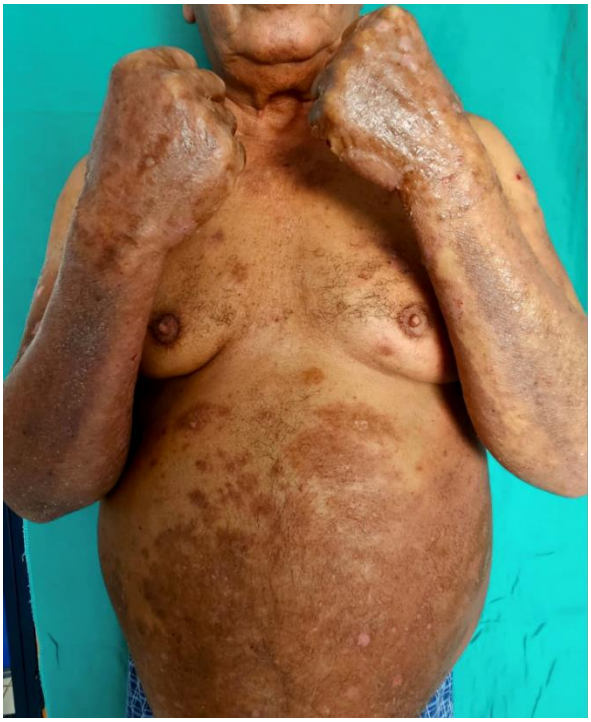

Figure 2 and 3: Bullous lesions on the Extensor surfaces of the limbs and the Abdominal area on a erythematosus background

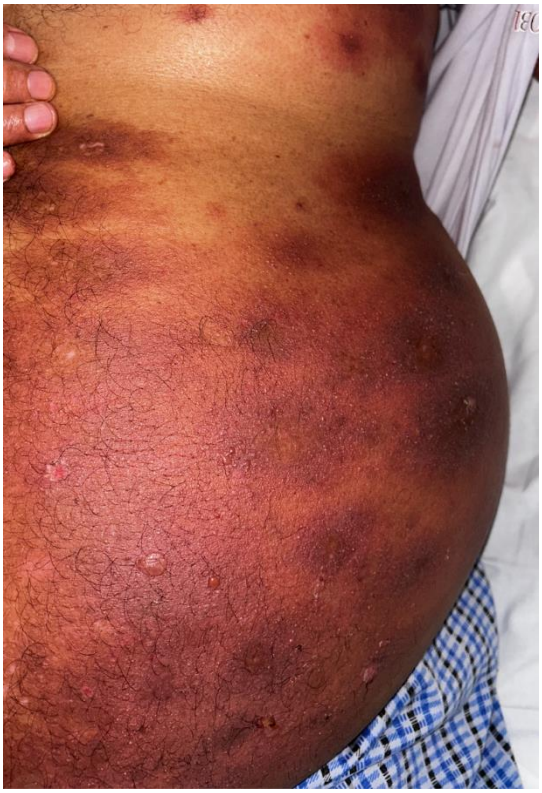

Figure 4: Close up of the bullous and post bullous erosions

DISCUSSION

Secukinumab is a fully human monoclonal antibody that selectively binds to interleukin-17A and prevents its interaction with functional receptors [3]. While highly effective and generally well-tolerated for moderate-to-severe psoriasis, cutaneous adverse events beyond standard injection-site reactions or routine infections are uncommon [4].

In this patient, several clinical features strongly point toward secukinumab as the primary culprit for drug-induced bullous pemphigoid. First, a clear temporal relationship was observed, as symptoms emerged within one month of reinitiating therapy. Second, all other potential causative medications were excluded, given that the patient received no concurrent systemic or topical treatments prior to or during secukinumab therapy. Third, a positive re-challenge occurred when the administration of a subsequent scheduled injection prompted an acute flare of the blistering eruption. Finally, definitive de-challenge led to complete clinical clearance upon permanent cessation of secukinumab.

Our observations align with similar rare reports in the literature. Guanghan and colleagues documented a 49-year-old male who developed bullous pemphigoid during secukinumab therapy for psoriasis, demonstrating no recurrence following drug withdrawal [4]. Isolated cases of bullous pemphigoid emerging during secukinumab treatment for psoriatic arthritis have also been reported [5].

Psoriasis pathogenesis is predominantly driven by T-helper 17 cell-derived cytokines, whereas bullous pemphigoid is heavily mediated by T-helper 2-associated cytokine pathways [6]. The precise mechanism by which interleukin-17A blockade induces subepidermal autoimmune blistering remains to be fully elucidated, but immune dysregulation or cytokine axis shifting following targeted biologic blockade may play a contributing role.

CONCLUSION

This case underscores the importance of recognizing secukinumab as a potential trigger for drug-induced bullous pemphigoid. Prompt clinical recognition, diagnostic confirmation via direct immunofluorescence, and immediate drug withdrawal—coupled with targeted anti-inflammatory regimens—are essential for effective patient management.

Source of Support: Nil

Conflict of Interest: none

REFERENCES

1. Karakioulaki, M., Eyerich, K., & Patsatsi, A. [2023]. Advancements in bullous pemphigoid treatment: A comprehensive pipeline update. *American Journal of Clinical Dermatology*, *25*[2], 195–212. https://doi.org/10.1007/s40257-023-00832-1 Cited by: 83
2. Krueger JG, Wharton KA, Schlitt T, *et al.,*IL-17A inhibition by secukinumab induces early clinical,

histopathologic, and molecular resolution of psoriasis. J Allergy Clin Immunol. 2019;144[3]:750–763. doi:10.1016/j.jaci.2019.04.029

3. Fotiadou, C., *et al.,*[2023]. Case Report: Coexistence of bullous pemphigoid and psoriasis—Dual therapeutic potential and paradoxes of IL-17A blockade. *Frontiers in Medicine*, 10, Article 1010884.
4. Wang, G., Hu, X., Han, S., & Zhou, M. [2024]. A case report of bullous pemphigoid following secukinumab therapy for a patient with psoriasis. *Journal of Dermatological Treatment*, *35*[1], Article 2366535. https://doi.org/10.1080/09546634.2024.2366535 Cited by: 5
5. Ho, P. H., *et al.,*[2021]. Development of bullous pemphigoid during secukinumab therapy for psoriasis: A case report. Dermatologica Sinica, 39[4], 209–210.
6. Zhang, L., Chen, Z., Wang, L., & Luo, X. [2023]. Bullous pemphigoid: The role of type 2 inflammation in its pathogenesis and the prospect of targeted therapy. *Frontiers in Immunology*, *14*, Article 1115083. https://doi.org/10.3389/fimmu.2023.1115083 Cited by: 41