Balkan Med J 2026;43:107-8

Secukinumab-Induced Interstitial Pneumonia in a Patient with Psoriasis

Siqi Li[1-3]*, Ru Dai[3]*, Qunye Xu[1,2]

[1]Department of Dermatology, Zhejiang University School of Medicine Second Affiliated Hospital Daishan Branch, Daishan, China
[2]Department of Dermatology, The First People's Hospital Daishan, Daishan, China
[3]Department of Dermatology, Second Affiliated Hospital, Zhejiang University School of Medicine, Hangzhou, China
*Contributed equally

Psoriasis is a chronic, relapsing, immune-mediated inflammatory skin disorder characterized by scaly, erythematous plaques. It significantly affects patients' physical and psychological well-being. Advances in medical research have led to the widespread use of biologic therapies targeting specific immune pathways, including tumor necrosis factor-alpha inhibitors, interleukin-12/23 (IL-12/23) inhibitors, and IL-17A inhibitors. Recent studies indicate that IL-17A inhibitors achieve higher absolute psoriasis area and severity index (PASI) response rates at 12 months in moderate-to-severe psoriasis compared with other biologicsin the treatment of moderate-to-severe psoriasis.[1] However, potential adverse reactions require careful monitoring to ensure safety and efficacy.

We report the case of a 65-year-old male with psoriasis vulgaris who developed interstitial pneumonia (IP) potentially induced by secukinumab treatment. The patient had a 10-year history of psoriasis, with no history of smoking or family history of autoimmune or respiratory diseases. He also denied prior interstitial lung changes or cancer. Initially, he received combined systemic and topical treatments, including oral glycyrrhizin tablets and topical corticosteroids, but these therapies were of limited efficacy. On physical examination, his PASI score was 10.3, body surface area involvement was 9%, and the dermatology life quality index (DLQI) score was 10 (Figure 1a). The patient was otherwise in good general health, with normal results for complete blood count, liver and renal function, myocardial function, and infection markers. A baseline chest computed tomography (CT) performed before initiating secukinumab showed no evidence of interstitial changes. Considering his medical history, secukinumab therapy was administered at 300 mg weekly

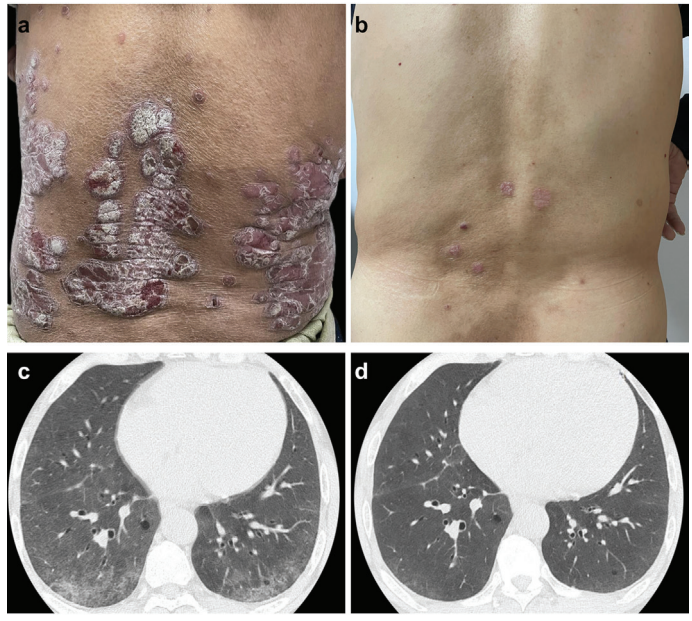

FIG. 1. Clinical and chest computed tomography (CT) course. (a) Multiple scaly erythematous plaques on the trunk. (b) Psoriatic plaques were mostly eliminated 1 year after secukinumab administration. (c) High resolution CT of the chest 1 year after the initiation of secukinumab therapy showing diffuse lamellar hyperdense shadows in both lungs, with multiple mesenchymal changes in the subpleural. (d) 10 months after the cessation of secukinumab therapy.

Corresponding author: Qunye Xu, Department of Dermatology, The First People's Hospital Daishan; Department of Dermatology, Second Affiliated Hospital, Zhejiang University School of Medicine, Daishan, Hangzhou, China

e-mail: 746009561@qq.com

Received: July 08, 2025 **Accepted:** September 06, 2025 **Available Online Date:** 02.02.2026 • **DOI:** 10.4274/balkanmedj.galenos.2025.2025-8-43

Available at www.balkanmedicaljournal.org

ORCID iDs of the authors: S.L. 0009-0005-1465-1909; R.D. 0000-0002-2691-8544; Q.X. 0000-0003-3496-7749.

Cite this article as: Li S, Dai R, Xu Q. Secukinumab-Induced Interstitial Pneumonia in a Patient with Psoriasis. Balkan Med J.; 2026; 43(2):107-8.

for the first 4 weeks, followed by 300 mg monthly. After 1 year of treatment, the patient's skin lesions were completely cleared, achieving a 90% improvement in PASI score, and the DLQI decreased to 5 (Figure 1b). However, follow-up CT revealed diffuse bilateral patchy opacities and subpleural interstitial abnormalities (Figure 1c). Serum KL-6 levels were markedly elevated at 2,759 U/mL. The patient remained asymptomatic, with no cough or sputum. Tests for human immunodeficiency virus, syphilis, hepatitis viruses, tumor markers, and antinuclear antibodies were negative. Bronchoscopy was unremarkable, but bronchoalveolar lavage fluid analysis showed a mildly increased lymphocyte proportion (10%). Based on these findings, a diagnosis of secukinumab-induced IP was established. Secukinumab therapy was immediately discontinued, and the patient did not receive antifibrotic treatment during this period. Follow-up chest CT demonstrated gradual resolution of the inflammatory changes 10 months after discontinuation (Figure 1d), and serum KL-6 levels decreased to 779 U/mL.

IL-17A is a cytokine involved in the pathogenesis of various autoimmune and inflammatory diseases. Secukinumab, a monoclonal antibody targeting IL-17A, is widely used for the treatment of psoriasis. While anti-IL-17A therapy has demonstrated benefits in reducing pulmonary fibrosis and inflammation in some studies, it has also been associated with paradoxical pro-inflammatory responses in other conditions, such as inflammatory bowel disease. KL-6, a glycoprotein secreted by type II alveolar epithelial cells, is markedly elevated in interstitial lung disease, primarily due to epithelial cell injury and subsequent regeneration. Serum KL-6 levels serve as a biomarker for disease activity and severity, showing a strong correlation with declines in pulmonary function and radiological progression.[2,3] A previous case reported a female patient with IP who experienced improvement in both lung disease and serum KL-6 levels during secukinumab treatment for psoriasis.[4] *In vivo* studies using mouse models of IP demonstrated that anti-IL-17A antibody treatment significantly suppressed lung inflammation and fibrosis.[5] Conversely, there have been five previous case reports of IP caused by secukinumab. Additionally, other IL-17 inhibitors, such as ixekizumab, have also been associated with IP.[6] Previous studies have identified risk factors for drug-induced IP in patients treated with anti-IL-17 biologics, including advanced age, elevated baseline KL-6 levels, and a prior history of IP.[7]

Although this is not the first reported case, the conflicting findings from previous studies on the impact of anti-IL-17 biologics on IP make this case particularly significant, providing new evidence that anti-IL-17A may contribute to the development of IP.

Informed Consent: The patient in this manuscript has given written informed consent to publication of his case details.

Authorship Contributions: Data Collection and/or Processing- Q.X.; Literature Review- S.L.; Writing- S.L.; Critical Review- R.D.

Conflict of Interest: No conflict of interest was declared by the authors.

REFERENCES

1. Pinter A, Eyerich K, Costanzo A, et al. Association of disease duration and PASI response rates at week 12 in patients with moderate-to-severe plaque psoriasis receiving biologics in the real-world psoriasis study of health outcomes (PSoHO). *J Dermatolog Treat.* 2024;35:2350227. [CrossRef]
2. Shiroshita A, Anan K, Shirakawa C, Shiba H, Yajima N. Longitudinal changes in KL6 levels predict acute exacerbation in fibrotic interstitial lung disease. *Sci Rep.* 2025;15:26061. [CrossRef]
3. Álvarez Troncoso J, Porto Fuentes Ó, Fernández Velilla M, et al. Evaluating the diagnostic and prognostic utility of serial KL-6 measurements in connective tissue disease patients at risk for interstitial lung disease: correlations with pulmonary function tests and high-resolution computed tomography. *BMC Pulm Med.* 2024;24:603. [CrossRef]
4. Miyachi H, Nakamura Y, Nakamura Y, Matsue H. Improvement of the initial stage of interstitial lung disease during psoriasis treatment with secukinumab. *J Dermatol.* 2017;44:e328-e329. [CrossRef]
5. Mi S, Li Z, Yang HZ, et al. Blocking IL-17A promotes the resolution of pulmonary inflammation and fibrosis via TGF-beta1-dependent and -independent mechanisms. *J Immunol.* 2011;187:3003-3014. Erratum in: *J Immunol.* 2014;193:5345-5346. [CrossRef]
6. Kaburaki S, Okada N, Tanaka T, et al. Ixekizumab-induced interstitial lung disease: a case report confirmed by transbronchial lung cryobiopsy. *J Dermatolog Treat.* 2024;35:2424338. [CrossRef]
7. Miyagawa H, Hara H, Araya J, et al. Characteristics of anti-IL-17/23 biologics-induced interstitial pneumonia in patients with psoriasis. *PLoS One.* 2021;16:e0245284. [CrossRef]