ABSTRACT BOOK ABSTRACTS



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ADVERSE DRUG REACTIONS, INCLUDING SJS, TEN

PARADOXICAL EXACERBATION OF LATENT INTERSTITIAL PNEUMONIA BY SECUKINUMAB IN A PATIENT WITH PSORIASIS VULGARIS

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Background: Recent clinical study showed anti-IL-17A antibody agent secukinumab (SEC) administration to psoriatic patients with increased KL-6 levels may suppress latent lung inflammation leading to reduced KL-6 level. In vivo studies using mouse model of IP demonstrated that anti-IL-17A antibody treatment significantly suppressed lung inflammation and fibrosis.

Observation: Here we report a unique case of psoriasis vulgaris treated with SEC, who paradoxically developed interstitial pneumonia. A 66-year-old male with psoriasis vulgaris was referred to our department. His psoriasis had been treated with topical corticosteroid, vitamin D3 and cyclosporine for 20 years. At initial presentation, multiple scaly erythematous plagues were scattered on the trunk and extremities. Blood tests revealed increased serum KL-6 value, 1408 U/mL (normal range <500 U/mL). Chest computed tomography (CT) showed slight grassy infiltration on the right lower lobe, in which radiologist considered nonspecific finding. Since skin atrophy was severe due to the long-term use of topical corticosteroids, we initiated SEC administration to reduce the need for treatment with topical agents. After its introduction, the patient's psoriatic plagues rapidly cleared. However, serum KL-6 level gradually increased further up to 4519 (U/mL). Ten months after the initiation of SEC, chest CT showed areas of mild reticular shadows in the subpleural area of the bilateral lower lobe. Since the concomitance of connective tissue disease and drug induced IP due to other than SEC were unlikely, he was diagnosed with SEC-induced IP. After the cessation of SEC, systemic corticosteroid was administered. The serum KL-6 level rapidly decreased and normalized. Follow-up chest CT showed reduced ground glass opacity 9 months after SEC cessation.

Key message: We speculated that slight lung inflammation of our patient was paradoxically exacerbated by SEC administration. The details of the paradoxical reaction to anti-IL-17A antibody agents are not clear. Further studies are warranted for elucidating its mechanism.





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