

Transition from secukinumab to adalimumab in COVID-19-Induced psoriasis flare-up treatment: a case report

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Abstract: Coronavirus disease 2019 (COVID-19) is known to trigger systemic inflammation and elicit immune responses, which may disrupt the delicate balance of cytokines involved in psoriatic regulation. Compared to other therapies in dermatology, biologics used for immune-mediated dermatological diseases have been more extensively studied during the COVID-19 pandemic. Herein, we report a case of flare-up of previously well-controlled psoriasis shortly after infection with COVID-19, with treatment transition from secukinumab to adalimumab.

Keywords: COVID-19; psoriasis; flare-up; adalimumab; secukinumab

Introduction

Psoriasis is a chronic immune-mediated skin disease that affects an estimated 125 million people worldwide^[1]. Existing literatures mainly reviewed psoriasis flares following COVID-19 vaccination, but few touched on flares among their patients' current therapy and flares after COVID-19 infection. Here, we first report a patient who developed worsen psoriasis shortly after COVID-19 infection, which was previously well controlled on secukinumab, and eventually successfully treated with adalimumab.

Case Report

A 62-year-old man, diagnosed as psoriasis vulgaris combined with coronary heart disease, had been well controlled on secukinumab (IL-17A inhibitor) since March 2022. Ten months later, the patient contracted the COVID-19 infection, but soon recovered in 2 days with a slight fever. However, erythema and scales gradually appeared on his trunk and limbs (Figure1. A-B), which showed no response to two consecutive doses of 300mg/month of secukinumab.

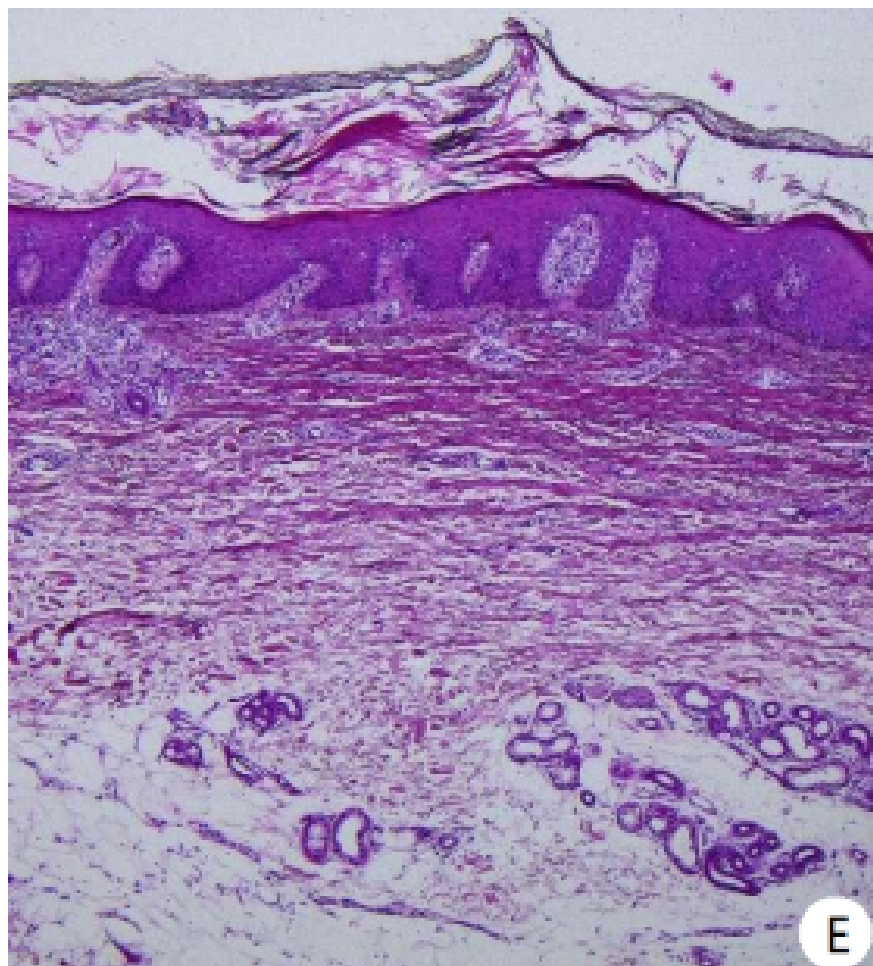
His blood tests revealed increased level of TNF, 9.98 pg/ml (normal value<8.10 pg/ml). While IL-6, C-reactive protein and erythrocyte sedimentation rate were normal. Skin biopsy (Figure1. E-F) indicated a psoriasiform dermatitis. Combining the patient's history of psoriasis, the disease was confirmed as psoriasis flare, and the rash subsided significantly after 8 weeks of adalimumab (TNF- α inhibitor) treatment (Figure1. C-D). The patient was still in follow-up.











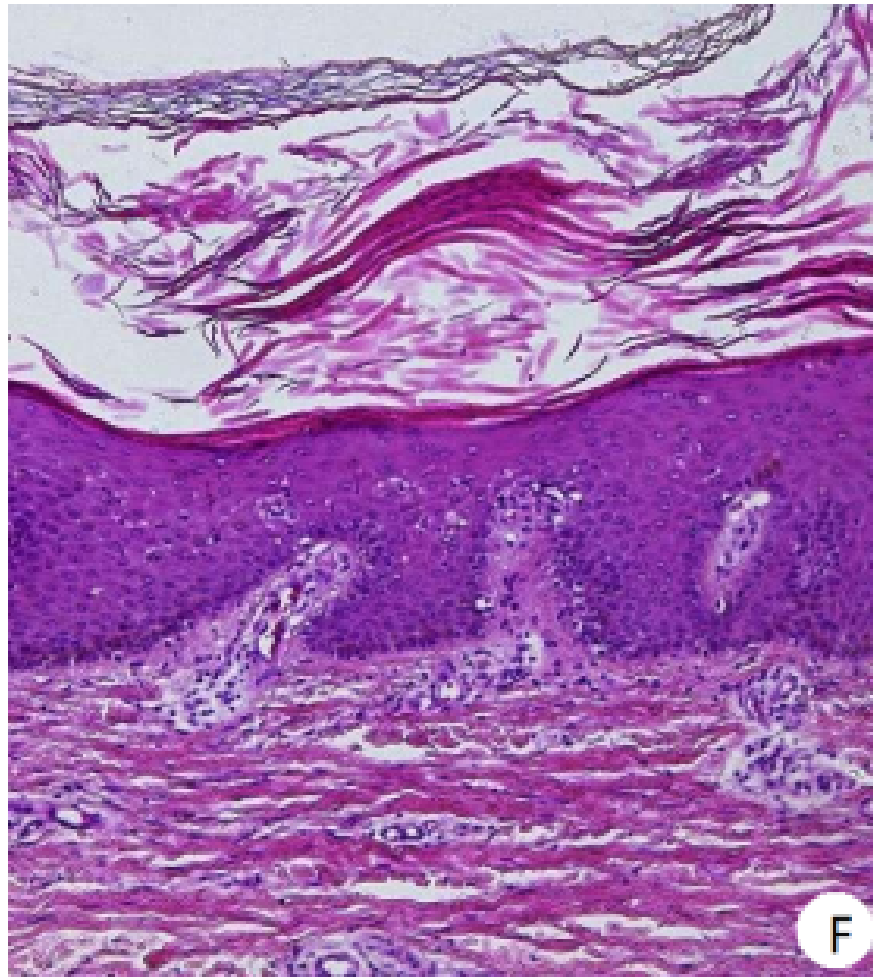


Figure 1. A-B. Cutaneous manifestations of the patient was noted by erythema and scales on his trunk and limbs. C-D. Cutaneous manifestations 8 weeks after starting adalimumab. E-F (Hematoxylin & Eosin stain, E:40×magnification; F:200×magnification). Skin biopsy was characterized by a greatly thickened epidermis with elongation of the rete ridges that protrude down into the dermis. There is retention of keratinocyte nuclei in the outer layers. These dermal blood vessels and surrounding upper dermal spaces are filled with predominantly mononuclear leukocytes and neutrophils.

Discussion

Viral infection has been shown to be strongly associated with the onset or exacerbation of psoriasis. The possible mechanism of COVID-19 infection leading to psoriasis flare-ups is the stimulation of toll-like receptor 3 by viral RNA, leading to dysregulation of the innate immune response and production of IL-36- γ and CXCL8. In addition, psoriasis may be exacerbated due to the hyperinflammatory state of COVID-19 patients^[2]. Our patient's recurrent rash was atypical, possibly related to the use of secukinumab therapy. Combined with the patient's past history and pathology, the diagnosis was psoriasis. Although the causality between the COVID-19 infection and psoriasis flare-up cannot be definitively established in this single case, previous study has reported associations between COVID-19 infections and the exacerbation of psoriasis^[3].

Currently, there is no consensus on whether biological agents increase the risk of coronavirus infection^[4]. Several studies have shown that the levels of circulating IL-17 are elevated in the peripheral blood of COVID-19 patients, therefore the use of IL-17 inhibitors may become a new treatment option for COVID-19, which

directly block the IL-17 pathway^[5]. That may also account for the mild symptoms of COVID-19 infection in our patient when using the treatment of secukinumab.

Despite the patient's previous positive response to secukinumab, the increasing psoriatic activity suggested possible resistance or tolerance to this IL-17A inhibitor. Blood tests of our patient revealed an increased level of TNF, a key cytokine in the process of psoriatic inflammation^[6]. The elevation of TNF may suggest a compensatory mechanism or an alternative pathway of psoriasis inflammation, warranting a change in the therapeutic approach.

In light of the lack of response to secukinumab and the increased TNF levels, a decision was made to switch the patient's treatment to adalimumab. The transition to adalimumab therapy led to a significant improvement, suggesting a relevant role of TNF in driving the psoriasis flare-up and reaffirming the importance of individualizing treatment choices based on patient characteristics and disease manifestations. The patient continues to be under follow-up to monitor the response to adalimumab and assess the long-term treatment outcomes.

It is essential to acknowledge the limitations of this case report. As a single-patient observation, it does not establish a causative relationship between COVID-19 infection and psoriasis flare-up or the efficacy of adalimumab. Further larger-scale studies, including controlled trials or observational studies, are needed to elucidate the complex interactions between viral infections and psoriasis, as well as to assess the comparative effectiveness of different biologic agents in similar scenarios.

In conclusion, this case highlights the potential impact of COVID-19 infection on psoriasis course and the importance of adapting treatment strategies to address individual patient responses. The patient's experience with secukinumab followed by adalimumab demonstrates the need for continuous evaluation and optimization of therapeutic approaches in psoriasis management, particularly in the context of viral infections or other triggering factors. Further research and clinical observations are warranted to develop a comprehensive understanding of the underlying mechanisms and to guide evidence-based treatment decisions in similar cases.

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