Comment on "Oral lichenoid reaction in a psoriatic patient treated with secukinumab: A drug-related rather than a class-related adverse event?"

To the Editor: We read with great interest the report by Capusan et al,[1] concerning an oral lichenoid eruption that developed in a psoriatic patient while treated with the interleukin (IL)-17A inhibitor, secukinumab (SEK). In that case, treatment discontinuation led to a complete resolution of the eruption, suggesting a causative role of the drug. Moreover, the eruption did not recur during treatment with the other IL-17 inhibitor, ixekizumab, allowing speculation that oral lichenoid reactions may represent a drug-related adverse event, rather than a class-related one.[1]

Recently, we reported a case of oral lichen planus triggered by infliximab-biosimilar CT-P13 that recurred when the patient was switched to SEK.[2] In our case, we did not observed a concomitant candida infection; however, during SEK treatment, the lichenoid eruption extended to the arms, trunk, and legs.

Mechanisms leading to paradoxical effects of biologics such as lichen/lichenoid reactions are still unknown. However, an increased release of interferon (IFN)-α by plasmacytoid dendritic cells, which are physiologically inhibited by tumor necrosis factor (TNF)-α, is believed to play a key role in anti—TNF-α—treated patients. Accordingly, experimental studies show that IFN-α is hyperexpressed in the lesions of patients with oral lichen planus.[3]

Because IL-17 and TNF-α have a synergistic effect and act in many inflammatory pathways in psoriasis, it is conceivable that IL-17 inhibitor—induced lichenoid reactions may recognize a similar pathogenic mechanism, centered on the activation of plasmacytoid dendritic cells.[4] Although currently there is no experimental evidence of this phenomenon, our observation could provide support for a common pathogenesis between IL-17 and TNF-α inhibitor—induced lichenoid eruptions and explain the recurrence of paradoxical reactions when shifting between biologics of the same or even different classes.

It is our opinion that, besides a genetic predisposition, the putative mechanism of increased release of IFN-α might be not enough alone to elicit the paradoxical reaction due to biologics, but other, maybe, exogenous factors, such as drug excipients or concomitant infections (even at a subclinical level), may play a triggering role as well. This finding might explain why in some patients the eruption appears only with a specific biologic or even spontaneously disappears without drug discontinuation, whereas in others the reaction recurs when shifting between biologics seemingly with different anti-inflammatory properties.

Although a biologic-induced paradoxical reaction does not represent a contraindication for other biologic treatments, clinicians must be aware of the risk of recurrence.

Antonella Di Cesare, MD, Roberto Maglie, MD, Leonardo Pescitelli, MD, Elia Rosi, MD, and Francesca Prignano, MD, PhD

Division of Dermatology, Department of Surgery and Translational Medicine, University of Florence, Florence, Italy

Drs Di Cesare and Maglie contributed equally to this work.

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Correspondence to: Francesca Prignano, MD, PhD, Department of Surgery and Translational Medicine, Division of Dermatology, University of Florence, Viale Michelangelo 41, 50125 Florence, Italy.

E-mail: francesca.prignano@unifi.it

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