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 observe 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.

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