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Vedolizumab-associated psoriasis: until where does gut selectivity go?

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Dear Editor,

The case was a 34-year-old female with A2L1B3 Crohn's disease diagnosed in 2005, with adalimumab introduced in 2011 that was interrupted six months later due to a thoracoabdominal herpes-zoster infection. Two months after adalimumab reintroduction, extensive and exudative scalp lesions appeared that were herpes-zoster-PCR negative. Adalimumab was stopped and topical-steroids were started, with a further worsening of exudative lesions that affected the entire occipital area with thick psoriasiform scales and a biopsy compatible with vulgar psoriasis. A complete resolution of skin lesions and intestinal disease remission was achieved with methotrexate, which was stopped in June 2017 due to pregnancy planning. Eight months later, systemic steroid-therapy was introduced due to a moderate/severe intestinal flare and vedolizumab was initiated in March 2018. After the 2nd infusion with ongoing steroid tapering, the patient only reported mild hair loss. Clinical remission was achieved in January 2019 without steroids and vedolizumab 300 mg 8/8 weeks was continued. In March 2019, the patient reported the reappearance of extensive scalp and peri-fistula psoriatic lesions. Topical therapy was started which was unsuccessful due to the progressive worsening of disabling lesions and

vedolizumab was suspended, with an improvement seen one month after discontinuation.

Discussion

Instead of *gut-selectivity*, few reports highlighted a *gut-focused* mechanism for vedolizumab, linking it to some dermatological manifestations such as psoriasis in IBD patients (1,2). A multicentric study reported the occurrence of skin adverse effects in 12 % of patients treated with vedolizumab, with this higher frequency explained by an immunosuppressive effect on target receptor expression outside the gut (3). Vedolizumab specific inhibition of α4β7-MAdCAM-1 binding occurs without affecting VCAM-1 binding, with evidence of Th1-cell differentiation and an increased number of FoxP3/Treg-cells in the skin (4). Thus, there may be a potential impact of vedolizumab on leukocyte cutaneous migration, with a local immunological unbalance similar to the effect of TNFα-inhibition on cytokine homeostasis as a trigger for psoriasis. This study highlights the potential of vedolizumab treatment to trigger severe paradoxical psoriasis in Crohn's patients, with a history of paradoxical psoriasis to anti-TNF. The underlying pathological mechanisms are largely unknown.

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