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Palmoplantar Pustulosis Induced by both Adalimumab and Golimumab for Treatment of Ankylosing Spondylitis

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Dear Editor:

Adalimumab is a fully humanized monoclonal tumor necrosis factor (TNF)-α antibody used to treat various inflammatory diseases, including ankylosing spondylitis, rheumatoid arthritis, and psoriasis[1]. Golimumab is an alternative TNF-α antagonist prescribed if other agents showed no effect, is associated with adverse events, or are unavailable[2]. Here, we report a case of palmoplantar pustulosis that developed after injections of both adalimumab and golimumab to treat ankylosing spondylitis.

A 52-year-old man presented with an asymptomatic skin rash on the palms and soles of 3 months in duration. He had ankylosing spondylitis, and was positive for HLA-B27. He had been treated with adalimumab (40 mg subcutaneously at 2-week intervals) for 3 years. He had no history of any other medical problem or dermatosis and his family history was unremarkable. On physical examination, erythematous scaly patches with pustules were evident on the palms and soles (Fig. 1). Histopathological examination revealed psoriasiform hyperplasia and intraepidermal bullae filled with neutrophils (Fig. 2). As adalimumab was thought to be the cause of the skin eruption, the rheumatologist decided to discontinue it. After 4 weeks of treatment with cyclosporine, topical calcipotriol, and a corticosteroid cream, the skin lesions improved. However, he suffered a relapse of back pain, so the adalimumab substitute golimumab (50 mg subcutaneously at 4-week intervals) was initiated to control the ankylosing spondylitis. After the fourth injection, the palmoplantar eruption recurred. As he needed to remain on a TNF-α antagonist, we decided to control the skin lesion with topical agents and occasional systemic cyclosporine and corticosteroids.

Psoriasiform eruptions are the most common paradoxical effects of TNF-α antagonists and can develop at any time from a few days to 4 years after drug initiation[1,3,4]. The pathogenesis remains unclear, but increased production of interferon-α by plasmacytoid dendritic cells, in which interferon synthesis is normally inhibited by TNF-α, is thought to play a role in development of the eruptions[1]. In addition, increases in the expression of chemokines and their receptors, with activation of the Th17 pathway, may be in play. In summary, the pathogenesis of psoriasiform eruptions caused by TNF-α antagonists seems to share a principal mechanism with that of idiopathic psoriasis[1].

In treatment, Collamer et al.[3] developed an algorithm. When such eruptions develop during TNF-α antagonist therapy, an infection should initially be ruled out. If the le-

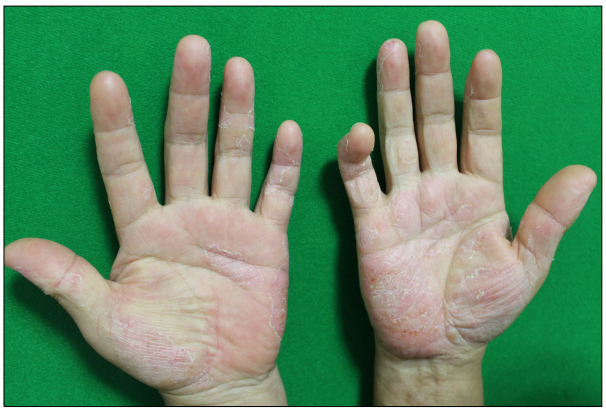

Fig. 1. Erythematous scaly patch with pustules on both palms were seen.

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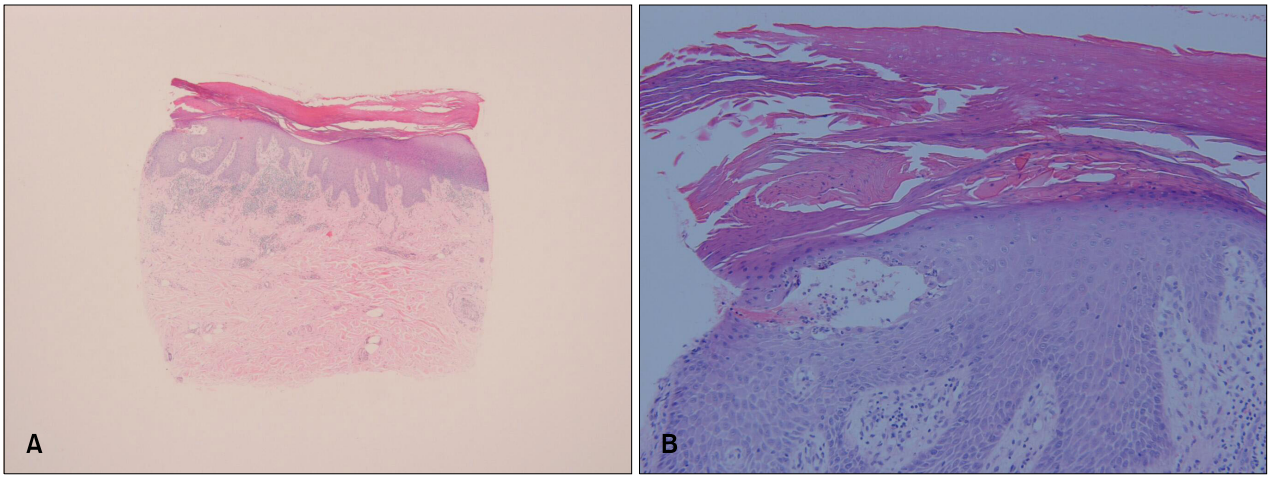

Fig. 2. (A) Histopathologic findings of the lesion showed psoriasiform epidermal hyperplasia with perivascular inflammatory cell infiltration in the papillary dermis (H&E, ×40). (B) Parakeratosis and intraepidermal spongiform bullae filled with neutrophils was observed in expanded view (H&E, ×200).

sion covers >5% of the body surface, or has palmoplantar psoriasis-like features (as in our case), an alternative TNF-α antagonist should be considered first. However, Nguyen et al.[5] found that switching the TNF-α antagonist did not adequately control the psoriasiform eruptions that develop paradoxically after treatment with TNF-α antagonists. In our patient, both adalimumab and golimumab induced palmoplantar pustulosis, and switching the TNF-α antagonist was not effective at controlling the condition.

We experienced a case of paradoxical palmoplantar pustulosis induced by both adalimumab and golimumab. We recommend that the TNF-α antagonist not be switched or stopped. Topical agents, treatments such as phototherapy, and systemic agents can be used to treat TNF-α antagonist-induced psoriasis in patients in whom autoimmune diseases are effectively controlled by TNF-α antagonists.

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