Psoriasiform Dermatitis Related with T-Cell Immunoreceptor with Immunoglobulin and Immunoreceptor Tyrosine-Based Inhibitory Motif Domains Inhibitor in a Patient with Non-Small-Cell Lung Cancer

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Dear Editor:

MK-7684, an antagonistic agent targeting T-cell immunoreceptor with immunoglobulin and immunoreceptor tyrosine-based inhibitory motif domains (TIGIT), is on phase 1 trial for the treatment of advanced solid tumors since December 2016. We present a case of psoriasiform dermatitis after treatment with MK-7684. Until date, there exists no reported case in English literature about psoriasiform dermatitis associated with TIGIT inhibitor.

A 63-year-old male with non-small-cell lung cancer (NSCLC) failed to respond to classic chemotherapy and was subsequently treated with pembrolizumab (Fig. 1A). After nine months, he developed erythematous plaques on his arms and legs without changes in his nails (Fig. 1B, C). The skin lesion was mild and controlled by topical steroids. Five months later, due to the progression of NSCLC, pembrolizumab was discontinued and MK-7684 was started subsequently. After one month, he developed eryth-

ematous scaly plaques on his whole body (Fig. 1D, E). He also developed trachyonychia on his twenty nails (Fig. 1F). He was a non-smoker and had been taking metformin and atorvastatin for more than two years. He denied a past medical history of psoriasis. After a skin biopsy, the lesion was histologically diagnosed as psoriasiform dermatitis (Fig. 2). He was treated with topical steroids and phototherapy. MK-7684 was stopped after the second injection due to exacerbation of psoriasiform dermatitis. No new lesions had developed after the discontinuation.

Drug-related psoriasis is a side effect of several drugs, such as NSAIDs, antimalarial agents, lithium, beta-blockers and anti-programmed cell death 1 (PD-1) antibodies¹. These drugs can induce psoriasis or exacerbate preexisting psoriasis¹. The time interval from drug administration to the appearance of the lesion varies from less than four weeks to three months or more¹.

TIGIT is a new checkpoint receptor target for cancer immunotherapy and thought to be upregulated on T-cells in multiple cancer models². By blocking TIGIT, the interaction of CD155 and CD112 with the costimulatory receptor CD226 can be enhanced in association with activation of T-cell mediated immune response against cancer cells³.

In our case, although mild psoriasiform eruption was induced during treatment with pembrolizumab, skin lesions progressed to biopsy-proven psoriasiform dermatitis during treatment with MK-7684. It has been hypothesized that over-activation of T-helper 1 and 17 cells through anti-PD-1 could contribute towards the development of psoriasis⁴. Although the pathological mechanisms of develop-

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Fig. 1. (A) Timeline of chemotherapy. (B) Erythematous plaques and papules during pembrolizumab treatment. (C) Normal nail plate during pembrolizumab treatment. (D, E) Hyperkeratotic scaly erythematous plaques during T-cell immunoreceptor with immunoglobulin and immunoreceptor tyrosine-based inhibitory motif domains (TIGIT) inhibitor treatment. (F) Nail dystrophy during TIGIT inhibitor treatment. We received the patient's consent form about publishing all photographic materials.

Fig. 2. Histopathological images of skin biopsy specimen of the leg. (A) Acanthosis with elongated rete ridges on epidermis and papillary dermal inflammatory infiltrations (H&E, $\times 100$). (B) Parakeratosis and microabscess in stratum corneum (H&E, $\times 400$).

ing psoriasiform dermatitis by TIGIT inhibitor are unknown, increased production of cytokines related to psoriasis could be one of the mechanisms⁵. Wang et al.⁵ reported about TIGIT+ CD4+ T-cells and cytokine levels in patients with psoriasis. The researchers reported that the activation of TIGIT inhibited psoriatic CD4+ T-cell proliferation, decreased the production of IFN- γ and IL-17A, and increased the IL-10 level⁵. They also reported an increase in the production of IFN- γ and IL-17A subsequent

to treatment with TIGIT inhibitor⁵. We believe that down-regulation of TIGIT on CD4+ T-cells might have contributed towards the pathogenesis of psoriasiform dermatitis in our case⁵. Therefore, we claim that clinicians should be aware of the potential development or aggravation of psoriasiform dermatitis when opting for TIGIT inhibitor treatment.

CONFLICTS OF INTEREST

The authors have nothing to disclose.

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Cutaneous Plasmacytosis Showing a Neuronal Involvement in a 35-Year-Old Female

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Dear Editor:

Cutaneous plasmacytosis (CP) is a rare skin disorder characterized by multiple reddish-brown patches and nodules that mainly occur on the trunk in adults¹. Histologically, there is a dermal infiltrate of mature plasma cells. We re-

port a case of 35-year-old female who presented with multiple red to brownish macules and patches on the left side of the trunk with a neuronal distribution (Fig. 1). The lesion appeared 4 years ago without any symptoms of pain or itching. A histopathological examination revealed a dense dermal patch-like cellular infiltration surrounding the follicles and the nerves (Fig. 2). The infiltrate was composed of lymphocytes, histiocytes, and plasma cells. Immunohistochemical staining was positive for CD138, CD3, and CD20. Serum protein electrophoresis and immunoelectrophoresis revealed polyclonal hypergammaglobulinemia. Based on these findings, a diagnosis of CP was made. The patient underwent treatment with systemic corticosteroids and narrowband ultraviolet B for 3 months till date. However, the response to the treatment has been minimal.

In our review of the literature, skin manifestations of CP were considerably uniform among patients, with multiple reddish to brownish colored scaly patches, nodules, and

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