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LETTER TO THE EDITOR

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A case of de novo psoriasis induced by atezolizumab in a patient with advanced lung cancer

Dear Editor

Atezolizumab is a humanized monoclonal antiprogrammed death-ligand 1 (PD-L1) antibody that started to be used internationally in 2016. It is indicated as a treatment for inoperable advanced or relapsed nonsmall cell lung cancer (NSCLC). Psoriasis may arise as an immune-related adverse event (irAE), is known to newly appear or be worsened after treatment with antiprogrammed death-1 (PD-1) agents, such as nivolumab.[1,2] However, there have been only a few reports on anti-PD-L1 agent-induced psoriasis.[3,4] Here, we report a case of de novo psoriasis induced by atezolizumab, which was successfully treated with the conventional therapy without discontinuation of atezolizumab.

A 70-year-old Japanese male with no history of psoriasis started to receive atezolizumab treatment (1200 mg/body, every 3 weeks) in July 2018 for advanced squamous NSCLC (cT3N1M0, stage IIIA), originating in the left upper lung. He had already received four courses of radiochemotherapy a year before, but had exhibited progressive disease. Pruritic scaly erythematous plaques appeared after the third round of treatment with atezolizumab and rapidly worsened (Figure 1A). Laboratory data were almost normal, except for an elevated serum squamous cell carcinoma (SCC) antigen level (6.0 ng/mL, normal range: ≤2.5 ng/mL), which was regularly measured as a tumor marker of lung cancer. Histopathologically, the lesion exhibited parakeratotic hyperkeratosis, elongated rete ridges, loss of the granular layer, perivascular infiltration of lymphocytes intermingled with eosinophils in the upper dermis, and capillary dilation in the dermal papillae (Figure 1B,C).

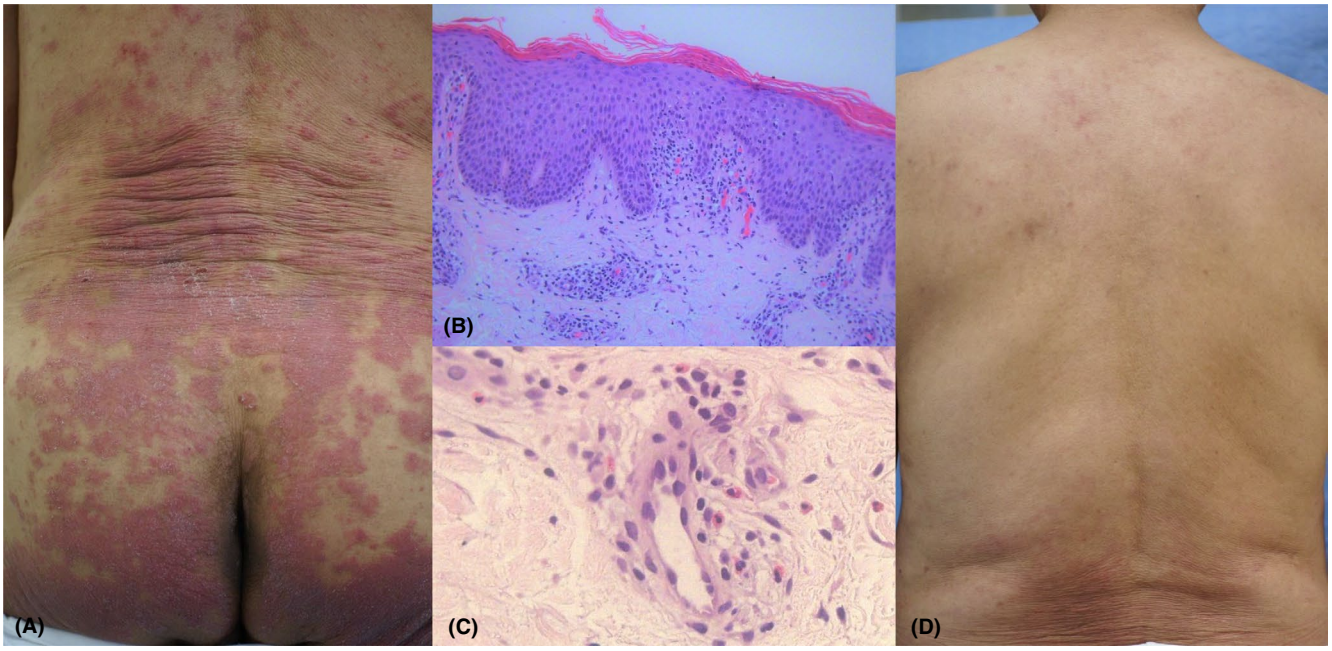

FIGURE 1 A, Well-demarcated, scaly erythematous plaques were seen on the trunk after the 5th round of atezolizumab treatment. B, A skin biopsy revealed parakeratotic hyperkeratosis, elongated rete ridges, loss of the granular layer in the epidermis, and capillary dilation in the dermal papillae (hematoxylin-eosin [HE] staining, ×200). C, Eosinophils infiltrated around the blood vessels in the upper dermis (HE staining, ×400). D, The skin rash almost disappeared after the 11th round of atezolizumab treatment

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We diagnosed the patient with de novo psoriasis, triggered by atezolizumab. The patient was treated with calcipotriol/betamethasone dipropionate combination ointment without discontinuation of atezolizumab treatment. The rash gradually improved after the 7th round of treatment, and it had almost disappeared by the 11th round of treatment (Figure 1D). The patient's serum SCC antigen level was changed in parallel with his psoriatic skin activity, peaking (18.1 ng/mL) between the 7th and 8th rounds of treatment. The patient noticed small lesions a couple of days after each treatment. His lung cancer was markedly ameliorated by the fourth round and exhibited a complete response at the time of the 19th round of treatment.

The blockade of PD-L1/PD-1 pathways has been demonstrated to augment T helper (Th) 1 and Th17 cell activity.[5] This results in increases of various psoriasis-related cytokines, such as interferon -γ, interleukin (IL) -17A, IL-21, IL-22, and IL-6 released from Th1 and Th17 cells.[6] Although little is known about which cytokines play an important role in the pathogenesis of anti-PD-1/PD-L1-induced psoriasis, Tanaka et al[7] reported that the serum level of IL-6 was increased in nivolumab-associated psoriasis and suggested that IL-6 might be a key cytokine in such irAE. However, it has not yet been clarified why our patient's skin lesions subsided despite continuous atezolizumab treatment.

Our case is the fourth reported case of anti-PD-L1-induced psoriasis[3,4] and the second case of atezolizumab-induced psoriasis. Among the previous cases, only our patient had no history of psoriasis and was successfully treated without discontinuation of the causative agent.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

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