Peripheral edema andurtain in a Patient treated with Atezolizumab

A 72-year-old man was referred to the dermatology clinic for evaluation of lesions after receiving his first dose of pembrolizumab (200 mg) for treatment of poorly differentiated adenocarcinoma of the prostate metastasis.

To the Editor:

Atezolizumab

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Atezolizumab (Genentech, South San Francisco, CA) is a humanized anti-programmed death-ligand 1 (PD-L1) monoclonal antibody approved for the treatment of urothelial carcinoma, non-small-cell lung cancer, and head-and-neck cancer. It is a humanized antibody to PD-L1 and is licensed for the treatment of non-small-cell lung cancer, urothelial carcinoma, and head and neck cancer with programmed death-ligand 1 (PD-L1) expression.

Atezolizumab is a humanized monoclonal antibody to PD-L1. It is used to treat patients with advanced urothelial cancer.

The use of Atezolizumab has been associated with various adverse reactions, including skin reactions such as contact dermatitis and paronychia.

The study was conducted in a patient with advanced urothelial cancer who was treated with Atezolizumab. The patient developed peripheral edema and urtication after the first dose of the drug. The edema was bilateral and resolved after discontinuation of Atezolizumab.

The authors conclude that Atezolizumab should be used with caution in patients with a history of dermatological adverse reactions, and that appropriate monitoring and management should be considered.

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A 72-year-old man was referred to the dermatology clinic for evaluation of lesions after receiving his first dose of pembrolizumab (200 mg) for treatment of poorly differentiated adenocar
was due to atezolizumab in only 1 case. In a recent series, 66% of patients had a previous history of psoriasis, which, in most cases, was controlled with topical treatment. Given the intensity of skin involvement, it was rarely necessary to suspend treatment or prescribe oral corticosteroids, as in the present case. In most cases, psoriasis is triggered after several doses. In the only case where psoriasis was triggered by atezolizumab, onset was after the first dose, as occurred in the present case.

In terms of etiology and pathogenesis, murine models have shown that PD-1 deficiency increases the likelihood of the psoriasis-like skin disease phenotype and that PD-1 can play a regulatory role in the development of the disease. Under normal conditions, the PD-1 pathway maintains normal immune homeostasis, which prevents autoimmune reactions or damage to healthy tissue. T-cell activation induced by PD-1 inhibitors—together with other factors—can contribute to development of psoriasis or exacerbations of existing psoriasis.

The low number of cases of psoriasis associated with atezolizumab is probably due to the mechanism of action, which spares PD-1 and PD-L2 binding. Owing to the different nature (IgG4 isotypes or IgG1 isotype), mechanisms of action, and antitumor action of anti-PD-1 and −PD-L1 agents, it has been recommended not to consider them as a group.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

References