
Please cite this article: S. Santín, J. M. Escrig, J. de la Concha.

Case of a 72-year-old patient with atezolizumab. A rare, severe cutaneous reaction.

A 72-year-old patient with atezolizumab was referred to the dermatology clinic with Sézary syndrome. The patient had received atezolizumab, an anti-PD-L1 monoclonal antibody, for the treatment of urothelial carcinoma since 2016. During this period, the patient developed a cutaneous eruption characterized by the appearance of red, pruritic, and painful plaques on the face, palms, and soles. The patient also experienced generalized pruritus and constitutional symptoms. The eruption worsened over time, and the patient was referred to the dermatology clinic for further evaluation.

The diagnosis of atezolizumab-induced cutaneous reactions was made based on the clinical presentation and the temporal relationship between the initiation of atezolizumab therapy and the onset of the eruption. The patient was immediately discontinued from atezolizumab therapy, and the symptoms began to resolve within a few days.

Please note that this case report is not intended to provide medical advice or guidance on the use of atezolizumab. Further research is needed to better understand the incidence and management of atezolizumab-induced cutaneous reactions.
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


J. Santos-Juanes, P. Munguia-Calzada, C. Álvarez-Fernández

a Servicio de Dermatología, Hospital Universitario Central de Asturias, Oviedo, España
b Servicio de Oncología Médica, Hospital Universitario Central de Asturias, Oviedo, España

* Corresponding author. E-mail address: jorgesantosjuanes@gmail.com (J. Santos-Juanes).

© 2018 Elsevier España, S.L.U. and AEDV. Published by Elsevier España, S.L.U. All rights reserved.