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Autoimmune Hemolytic Anemia as a Complication of Atezolizumab (Anti-PD-L1)

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ABSTRACT

Immune checkpoint inhibitor atezolizumab is currently FDA approved for the treatment of locally advanced or metastatic transitional cell carcinoma (TCC). Exceptionally, it can induce adverse effects related to the immune system. AIHA is a rare complication of atezolizumab but it must be taken into account because secondary effects related to the hematopoietic system have been recently reported.

Given the current and increasing indications for these drugs, it is essential for all physicians to become well versed with their common adverse effects in order to address early detection and better management.

We report a case of autoimmune hemolytic anemia in a patient with CCT and lymphatic node treated with atezolizumab.

Keywords: Atezolizumab, Programmed death receptor 1 (PD-L1), Autoimmune hemolytic anemia (AIHA)

INTRODUCTION

Atezolizumab is a humanized monoclonal antibody that belongs to the family of checkpoint inhibitors. It can bind directly to the ligand of the programmed death receptor 1 (PD-L1), providing PD-L1/PD-1 mediated inhibition of the immune response and reactivation of the antitumor response without inducing the cell-dependent cytotoxicity of antibody. Currently, it is approved for the treatment of locally advanced or metastatic transitional cell carcinoma (TCC). Exceptionally, it can induce adverse effects related to the immune system such as pneumonitis, colitis, pruritus and hepatitis. Recently, secondary effects related to the hematopoietic system have been reported, such as autoimmune hemolytic anemia (AIHA).

OBJECTIVE

To present a case of AIHA in a patient with CCT under treatment with Atezolizumab.

CASE REPORT

A 57 year old woman with CCT and lympatic node affectation diagnosis in November 2017 who started chemotherapy (CDDP-gemcitabine) in April 2018. After performing the last cycle of first-line chemotherapy (July 2018), persistence of hyper metabolic lymph nodes was evidenced, so cystectomy and lymphadenectomy were rejected. Three months later, given the adenopathic progression, it was decided to start treatment with Atezolizumab. One week after the third cycle (January 2019), the patient went to the Emergency Department with syncope, presenting hemoglobin (Hb) of 5.1 mg/dL that required transfusional support. The pre-transfusion tests showed: positive irregular antibody screening, nonspecific agglutination for a panel of 11 cells with presence of positive self-control, direct positive Coombs test (polyspecific 2+, IgG 2+ and negative Cd3) and eluted with nonspecific panglutination. After treating the sample with PEG, the presence of alloantibodies was ruled out. An erythrocyte genotyping was also performed using BloodChip technology. Finally, in accordance with the results obtained, it was transfused with isophenotype blood and negative cross-tests. The analytical results founded were compatible with an AHAI (reticulocytes: $274.3 \cdot 10^9$/L, haptoglobin <8.06 mg/dL, lactate dehydrogenase: 948 U/L, bilirubin 5.8 mg/dL).

Due to the absence of other causes and the recent administration of the drug, AIHA was suspected as a secondary complication of the anti-PD-L1. The Atezolizumab interruption and initiation of corticosteroid

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therapy was performed. The patient did not show the expected response with a persisting anemia, even though without clinical or hemodynamic repercussion, so no more transfusions were required. Treatment with infusion of Rituximab (MAb anti-CD-20) weekly at a dose of 375 mg/m^2 for 4 weeks was decided.

CONCLUSION

AIHA is a rare complication of Atezolizumab but it must be taken into account because it can put the patient's life at risk. Further research and documentation on this manifestation would be justified in order to address early detection and better management.

In our case, given the suspicion of AIHA due to Atezolizumab, it was decided to change the treatment to Rituximab. After three infusions the patient presented a notable analytical recovery (Hb: 10.5 mg/dL).

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