

Atezolizumab-induced autoimmune diabetes mellitus presenting with diabetic ketoacidosis

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Background: Atezolizumab is a humanized monoclonal, anti-programmed death ligand 1 (PD-L1) antibody used for the treatment of various cancers including metastatic breast cancer. Although immune checkpoint inhibitors are generally well tolerated compared with traditional chemotherapy, they can produce immune-related adverse events, including autoimmune diabetes. Autoimmune diabetes has rarely been seen with anti-PD1/PD-L1 therapies such as atezolizumab. In this report, we describe a patient with a known history of early breast cancer who presented with diabetic ketoacidosis (DKA) following adjuvant atezolizumab treatment.

Case Presentation: A 33-year-old women with early triple negative breast cancer received adjuvant 1000mg/m² capecitabine and 1200mg atezolizumab treatment every 3 weeks. She had history of type 2 diabetes mellitus and 2nd degree AV block (Morbitz type I), and no history of autoimmune conditions. On day 4 of second cycle of atezolizumab, she visited the emergency room on polydipsia and mental change who presented with DKA symptoms. In arterial blood gas analysis, the pH was 7.14 and the bicarbonate was 3.1 mmol/L, indicating severe metabolic acidosis. Hemoglobin A1c (HbA1c) was 10.1% with random serum glucose levels ranged from 150 to 300 mg/dl. And suppressed fasting C-peptide was 0.32ng/ml, borderline-low fasting insulin level was 1.0μIU/ml. Her initial management included intravenous sodium bicarbonate administration and insulin pump infusion, but metabolic acidosis with confused mentality was not resolved. Brain MRI was performed to differentiate autoimmune encephalopathy, but there were no abnormalities. Due to refractory metabolic acidosis, intermittent hemodialysis applied at third day of hospitalization. After treatment for DKA, she recovered mental status, and received multiple daily insulin injection therapy and oral hypoglycemic agents. Atezolizumab has been discontinued after the second cycle. After 2 months later, her HbA1c level declined to 8.3%.

Conclusion: PD-L1 inhibitor can induce autoimmune DKA. Regular monitoring of blood glucose levels and related symptoms in patient using PD-L1 inhibitor is the best way to detect DKA early.

