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Checkpoint inhibitor treatment induces an increase in HbA1c in nondiabetic patients

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Abstract

Immunotherapy greatly improves clinical outcomes in treated patients with cancer. However, the long-lasting immune response and long duration of therapy could induce long-term adverse effects owing to the chronic inflammation induced. Type 2 diabetes is now recognized as an inflammatory disease. In addition, immunotherapy is concerned with increase in the production of tumor necrosis factor-α, interleukin-2, and interferon-γ, which are involved in the inflammatory process. Based on these observations, we hypothesized that anti-programmed cell death-1 (anti-PD-1) and/or anticytotoxic T-lymphocyte-associated protein-4 therapy could contribute to type 2 diabetes genesis in treated patients. Therefore, to evaluate this hypothesis, we studied HbA1c levels during follow-up in patients treated with anti-PD-1 and/or anticytotoxic T-lymphocyte-associated protein-4 therapy. A prospective and observational study was performed in an oncodermatology department (Saint-Louis Hospital, Paris, France) from March 2015 to February 2017. Sixty-two patients meeting the inclusion criteria were enrolled. Forty-three patients had paired HbA1c measurements during their follow-up period and were analyzed. The median follow-up was 3 months. We noted an increase in HbA1c levels from 5.3% [interquartile range (IQR): 5.1-5.5; range: 4.5-6.2) to 5.45% (IQR: 5.2-5.7; range: 4.7-6.2; P=0.037). This observation was confirmed in the subgroup of patients who did not receive concomitant glucocorticoids; their median HbA1c levels increased from 5.3% (IQR: 5.1-5.5; range: 4.7-6.2) to 5.5% (IQR: 5.2-5.7; range: 4.7-6.3; P=0.025). Variables such as age, BMI, and sex were not associated with the HbA1c level increase, but a tendency toward rising HbA1c levels was observed in treatments longer than 12 months. This study demonstrates that treatment with anti-PD-1 antibodies may impair glucose metabolism, as measured by increasing HbA1c levels.

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