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CONGRESS

CORE BINDING FACTOR ACUTE MYELOID LEUKAEMIA FOLLOWING IMMUNE CHECKPOINT INHIBITION FOR SOLID TUMOURS: TWO CASE REPORTS AND LITERATURE STATE OF THE ART

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Background and Importance

Immune checkpoint inhibition (ICI) can induce responses in patients with advanced malignancies. Although a well-established downside of ICI is its diverse spectrum of immune-related adverse events, **the incidence of second primary malignancies associated with ICI is still a matter of debate.**

Materials and Methods

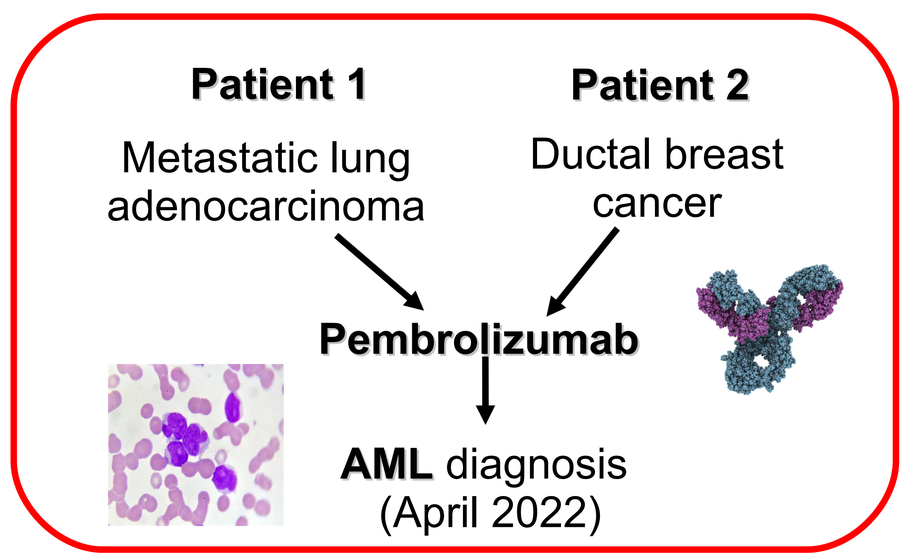
In both Patients 1 and 2, peripheral blood (PB) and bone marrow blood testing confirmed Core Binding Factor (CBF) AML, according to the presence of (inv16)(p13;q22) in 80% and 70% of blasts in the PB, respectively.
According to ESMO AML Guidelines, therapy with gemtuzumab ozogamycin associated with standard chemotherapy was recommended for both patients.

Results

Patient 1 achieved a CR after induction and consolidation therapy; patient 2 performed cytarabine-based consolidation therapy due to leukemia-aberrant immunophenotype. **At current follow-up (9 months after diagnosis) both patients are alive: in patient 1 negative CBF was confirmed and patient 2 had PD treated with the association azacitidine + venetoclax.**

Aim and objectives

We present two consecutive patients treated in our Hospital in 2022 who developed clinically acute myeloid leukemia (AML) during or after ICI treatment for solid tumors.
Patient 1 is a man with a previous history of metastatic lung adenocarcinoma treated with pembrolizumab, which was stopped due to complete response (CR) 5 months before diagnosis of AML in April 2022.
Patient 2 is a woman, with a previous history of ductal breast cancer treated with adjuvant chemoradiotherapy; she also developed a metastatic V600E BRAF-mutated melanoma, treated with BRAF/MEK inhibitors. Finally after two months of pembrolizumab, she developed AML in April 2022.



Conclusion and Relevance

A case of AML after 3 cycles of pembrolizumab for the treatment of non-small cell lung cancer and 5 cases of mieloyd neoplasia after treatment with ICIs were recently reported.
Hyperprogression of subclinical myeloid malignancies could be a potential explanation, since a myeloid clone with acquired driver mutation(s) could obtain an extra proliferation advantage from functional myeloid PD-1 knockout after ICI. Abberant PD-1 expression was observed in 8–26% of CD34+ blasts in myelodysplastic syndromes, chronic myelomonocytic leukemia and AML.
Moreover chemotherapy and BRAF inhibitor exposure, together with short exposure to pembrolizumab in Patient 2, suggest a major role of previous therapies in the development of AML.
The correlation between ICI and myeloid neoplasias is still uncertain.

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References and/or Acknowledgements

van Eijs, M.J.M.,et al. Cancer Immunol Immunother (2022)