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CML With MPAL Blast Crisis: A Therapeutic Conundrum Managed With FLAG/GO And Blinatumomab

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Abstract

Background

Blast-phase transformation of chronic myeloid leukemia (CML) carries a poor prognosis, and mixed phenotype acute leukemia (MPAL) transformation is rare. MPAL is a lineage-ambiguous, high-risk leukemia with no consensus treatment and poor outcomes representing a critical unmet need. Herein we report a patient managed successfully with FLAG/GO induction followed by blinatumomab plus ponatinib consolidation.

Methods

A 30-year-old woman presented with abdominal pain for 3 days and was incidentally found with a WBC count of 160,000/μL, platelets 1.8 × 10^6/μL and positive BCR::ABL1 mutation without excess blasts confirming CML in chronic phase. She was started on imatinib achieving a good response in her cell counts and decreasing BCR::ABL1 transcripts. Two months later she developed elevated transaminases, liver biopsy confirmed drug-induced liver injury, with imatinib and allopurinol as the leading candidate agents, prompting discontinuation of both agents. She was switched to asciminib, but remained on and off therapy because of intolerance and non-adherence. On routine follow-up she was found incidentally to have 65% circulating blasts, consistent with blast phase transformation. Bone marrow biopsy confirmed B/myeloid MPAL, with an ASXL1 mutation and inv(16). Given the myeloid-predominant, CD33-expressing disease with core-binding-factor inv(16), she received one cycle of FLAG/GO induction. Induction course was complicated by septic shock secondary to E. coli bacteremia, a self-limited trace subarachnoid hemorrhage, and transfusion-refractory pancytopenia. Post-induction bone marrow biopsy after count recovery revealed complete remission (CR). She declined to undergo allogeneic HCT and was started on blinatumomab plus ponatinib consolidation, leveraging CD19 expression on the pre-induction lymphoblast population. She achieved a deep molecular response (MR4.5) after two cycles of consolidation. After four consolidation cycles, she will proceed to blinatumomab-POMP maintenance.

Conclusion

Our patient with MPAL transformation of CML had a successful treatment from FLAG/GO induction followed by deepening molecular response with a chemotherapy-free, TKI based consolidation incorporating blinatumomab. While it is difficult to determine whether blinatumomab contributed independently to the deepening response over ponatinib alone; nevertheless, this case underscores the therapeutic complexity of

blast-phase CML manifesting as MPAL. The co-occurrence of core-binding-factor inv(16), an ASXL1 mutation, and a mixed B/myeloid immunophenotype illustrates how lineage plasticity can complicate both classification and treatment selection. Our approach of treating her with FLAG/GO induction exploited the CD33 expression and the favorable biology of inv(16), while blinatumomab plus ponatinib consolidation leveraged CD19 positivity on the lymphoblast population, allowing a potent yet chemotherapy-sparing treatment option in a patient who had already demonstrated significant treatment intolerance and declined allogeneic stem cell transplant.

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