Sequential Development of CLL in a Patient with CML Treated with Nilotinib: A Case Report

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Introduction

Coexistence of chronic myeloid leukemia (CML) and chronic lymphocytic leukemia (CLL) in the same patient is exceptionally rare. These are two distinct clonal hematologic malignancies with differing pathogenesis and treatment strategies.

Patient

52-year-old male presented with a one-month history of left hypochondrial pain. Physical examination revealed splenomegaly extending four fingers below the costal margin.

Diagnosis

Complete blood count showed marked leukocytosis

- Total leukocyte count (TLC) 210 × 10^9/L
- Hemoglobin 10.2 g/dL
- Platelets 239 × 10^9/L.

Blood film demonstrated a picture of CML in chronic phase. Quantitative PCR for BCR::ABL1 confirmed the diagnosis of CML in chronic phase, with a BCR::ABL1 transcript level of 73%. The sokal score was intermediate.

Outcomes

The patient was initiated on Nilotinib and achieved major molecular response (MMR), with BCR::ABL1 levels dropping to 0.035% after 6 months.

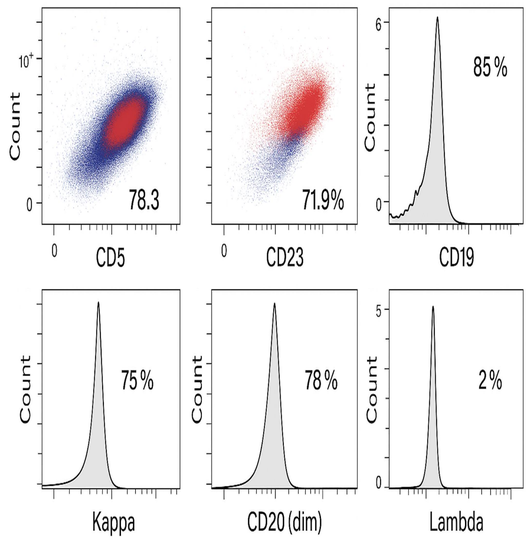
Rising leukocytosis with a TLC of 20 × 10^9/L was noted during follow-up after 3 years despite molecular remission.

Flow cytometry revealed a monoclonal B-cell population consistent with CLL.

Fluorescence in situ hybridization (FISH) testing showed no 17p deletion.

The patient remained asymptomatic with no cytopenias or bulky disease and was not indicated for CLL-directed therapy.

The patient is still in MMR on Nilotinib.



Flow cytometric immunophenotyping of peripheral blood leukocytes revealing a monoclonal B-cell population co-expressing CD5, CD19, CD20 (dim), and CD23, with restriction to kappa light chain, consistent with CLL immunophenotype.

Summary

We describe a rare case of sequential CLL arising in a patient with CML who was being successfully treated with nilotinib. This case highlights the need for vigilance for secondary hematological malignancies in patients on long-term TKI therapy.

The co-occurrence of CML and CLL remains an uncommon clinical scenario, and its pathogenesis is not fully understood. It may represent a chance convergence of two common leukemias, or there may be facilitating factors such as treatment-related immunomodulation or genetic predisposition.

In our patient, the emergence of CLL did not necessitate immediate therapy and could be managed conservatively alongside ongoing CML treatment.

Further studies and accumulated case experiences are needed to clarify whether TKI therapy can contribute to secondary lymphoproliferative disorders and to determine the best management practices for patients with dual leukemias

References

Saowapa S, Pangkanon W, Adu Y et al. A rare case of an elderly male with progression to chronic myeloid leukaemia secondary to chronic lymphocytic leukaemia. *EJCRIM*. 2024;11(4).

James D, Green S, Molica S, Allsup D. High-Risk Chronic Lymphocytic Leukemia complicating the course of imatinib-treated chronic myeloid leukemia: successful disease management with dual tyrosine kinase inhibition. *Case Reports in Hematology*. 2024 Nov 15;2024:1813512.

Parikh SA. Chronic lymphocytic leukemia (CLL) in the setting of chronic myeloid leukemia (CML): an uncommon coexistence. *Blood Cancer Journal*. 2013;3(3):e110.

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