P1.08 Multiparameter flow cytometric analysis of composite lymphoma; case report of a mantle cell lymphoma associated with a B-cell chronic lymphocytic leukemia and an aberrant T cell subset

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Introduction

Composite lymphomas (CL) are characterized by the presence of two or more simultaneous and clonally distinct lymphomas and may account for up to 5% of patients with chronic lymphoproliferative diseases. Identification of CL is facilitated by the use of 8- to 10-colour immunophenotyping, allowing the study of multiple antigens across different populations. Integration with morphology, cytogenetics and molecular analysis is necessary to distinguish CL from a single lymphoma clone in transformation.

Case report

A 68-year-old woman with a history of breast adenocarcinoma and rheumatoid arthritis presented with a differential white blood cell count comprising atypical lymphocytes. The absolute lymphocyte count was slightly elevated at 6620/mm³. There was no evidence of adenopathy, organomegaly or B symptoms. Flow cytometry analysis was performed on peripheral blood and revealed four distinct abnormal lymphocyte subpopulations:

- 1% of total leucocytes (TL) expressed the abnormal T cell phenotype CD3+ CD4+ CD5+ CD7+ CD2+ CD8-.
- 8% of TL (816 cells/µl) were of CD5+ classical B-CLL phenotype with monoclonal kappa light chains and a Maturity score of 5 points.
- 18% of TL (1836 cells/µl) exhibited a CD5+ Kappa+ phenotype compatible with mantle cell lymphoma (MCL).
- 11% of TL (1122 cells/µl), not expressing CDS antigen, were monoclonal Kappa+ compatible with an MCL blastoid clone.

Both MCL and CLL clones were detected in the bone marrow. Molecular analysis of Immunoglobulin rearrangement confirmed the Kappa monoclonality of B cells. IgH/Cyclin D1 rearrangement t(11;14) was not detected while SOX11 was overexpressed, which confirmed the diagnosis of MCL. FISH analysis reported trisomy 12, the second most common chromosomal abnormality in B-CLL.

Conclusion

The use of multi-colour flow cytometry is critical to identify several coexistent clones with aberrant phenotypes and, in this case, provides coherent data with the simultaneous presence of trisomy 12 and overexpression of SOX11. Detailed cytological and phenotyping data will be shown.