

Conference Abstracts

Vol. 6, Issue Supplement 2, 2026

Epidemiologic Characterization of Risk Factors in Patients with Dual Diagnosis of B-cell and T-cell Lymphomas

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Keywords: B-cell lymphoma, T-cell lymphoma, dual-lineage lymphoma, secondary lymphoid malignancy, bidirectional risk, Epidemiology

Submission received: 2026-08-15 / Accepted: 2026-08-25 / Published: 2026-09-25

<https://doi.org/10.53876/001a.129827>

Abstract

Background

Epidemiological studies demonstrate a nearly fivefold bidirectional risk between B-cell lymphomas (BCL) and T-cell lymphomas (TCL); however, the mechanisms underlying dual diagnoses remain poorly understood. Characterizing this population may provide insight into shared risk factors and mechanisms of lymphomagenesis.

Methods

We retrospectively analyzed 112 adult patients with dual BCL and TCL diagnoses from separate biopsy specimens at MD Anderson Cancer Center between 2013 and 2026. Patients with composite BCL and TCL were excluded. Clinical and demographic characteristics, including lymphoma subtype, T-cell receptor rearrangement (TCRr), non-lymphoid malignancies (NLMs), autoimmune disorders (AD), family history, and viral serologies, were collected.

Results

Among 112 patients, 61% were male. Median age at first and second lymphoma diagnosis was 63.5

(range, 18–86) and 67 (range, 19–88) years, respectively. BCL was the initial diagnosis in 66.7% of patients. TCRr confirmed clonality in 80.3% of tested TCL specimens. NLMs occurred in 37.5% of patients, with 35.7% having >1 NLM. Myeloid neoplasms accounted for 11.9%, while non-melanoma skin cancers represented the majority of NLMs. AD occurred in 15.2% of the cohort, most commonly rheumatoid arthritis and psoriasis. Family history of hematologic malignancy occurred in 16.1% of patients. HIV serologies were negative in all patients, whereas Hepatitis C, Hepatitis B, and CMV seropositivity occurred in 1.2%, 11.5%, and 60.3%, respectively, of those with data available. EBV-ISH was positive in 13.8% of BCL tumors and 22.3% of TCL specimens, with concurrent positivity in 4.2%.

Conclusion

Patients with dual BCL and TCL diagnoses had high frequencies of additional malignancies, autoimmune comorbidities, EBV positivity of tumors, and family history of malignancy. These findings suggest immune dysregulation as a potential shared predisposing factor alongside

environmental and genetic susceptibilities. explore underlying mechanisms of dual B-cell
Genomic and molecular analyses are required to and T-cell lymphomagenesis.

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