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Title

BCL2 overexpression in diffuse large B-cell Lymphoma

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Introduction

CL-2 family proteins are regulators of apoptosis, their overexpression in de novo DLBCL patients is associated with a poor prognosis. This pathway has already been used as a therapeutic target in the fight against cancer. Anti-BCL2 therapies (Venetoclax) are now a standard treatment for acute myeloid leukemia. In DLBCL, anti-BCL2 is already being evaluated for first-line treatment in combination with R-CHOP in BCL2+ cases. In this study, we aim to identify the clinical, epidemiological, and prognostic features of our BCL2+ DLBCL patients.

Methodolgy

This is a retrospective, descriptive, and analytical study, from January 2018 to December 2021, and includes newly diagnosed patients with BCL2+ DLBCL during this period. Data were collected using a computerized system (HOSIX). Statistical analysis was performed with IBM SPSS Statistics. Survival analyses with corresponding P-values were calculated using the Kaplan-Meier method.

Results

among a total of 184 DLBCL patients, 36 patients (19.56%) were BCL2+, consisting of 21 women and 15 men with a F/M sex ratio of 1.4. The median age at diagnosis was 56.77 years [19-81 years], and the average diagnostic delay was 5.9 months. Half of the patients had B signs at diagnosis, and 26 (72.2%) had a tumoral syndrome (adenopathy and/or splenomegaly).

Performans status was ≤1 in 30 patients (83.3%). Histological examination showed a proliferation index greater than 80% in 16 patients (44.44%), CD23 positivity in 4 patients (12%), and BCL6 positivity in 13 patients (36.2%). CT-Scan showed extensive involvement (stage III/IV) in 23 patients (63.9%). A bulky mass was described in 16 patients (44.4%). The IPI score was greater than three in 12 patients (39.9%). Four patients developed venous thrombosis.

Complete blood count revealed anemia in 30.55% of patients, lymphopenia in 16.6% of patients, thrombocytopenia in 8.33% of the patients, and neutropenia was observed in only one patient.

Thirty-one patients were placed on the RCHOP protocol. Primary mediastinal DLBCL cases were given a dose-adjusted R-EPOCH regimen. Unfortunately, three patients died before initiation of therapy. Twenty-one patients (63.6%) achieved remission after first-line therapy. Seven patients were declared refractory, and we lost three patients during follow-up.

We found a significant correlation between BCL2 overexpression and BCL6 positivity ($p=0.026$) and the refractory character ($p=0.045$). The positivity of BCL2 was not significantly associated with death ($p=0.218$). The 4 year-survival of our BCL2+ patients is 64% ($p=0.463$).

Conclusion

This poor prognosis and higher risk of non-response must be taken into account in the guidelines. The BCL2 marker will enable better stratification of DLBCL lymphoma patients and offer them more effective first-line treatment options. In theory, the future is bright for these patients, yet access to drugs may well be our toughest challenge in the years ahead.