

OS. Similarly, age ≥ 60 years (HR 1.08, $p < 0.001$), involvement of ≥ 4 nodal areas (HR 1.27, $p < 0.001$), and high LDH (HR 1.65, $p = 0.002$) predicted decreased PFS. Serous effusions (HR 2.09, $p = 0.05$), albumin < 3.5 g/dL (HR 2.82, $p = 0.05$), B-symptoms (HR 2.00, $p = 0.006$), involvement of ≥ 4 nodal areas (HR 1.21, $p = 0.014$), and BM infiltration (HR 2.11, $p = 0.05$) were the main predictors for HT. **Conclusion:** Although it is characteristically an indolent disease, we demonstrated that a significant portion of FL patients have shortened survival. We confirmed this prognostic heterogeneity, particularly considering the clinical staging and tumor burden. Therefore, FL patients with AS-HTV had higher HT rates and early-relapses, both classic adverse prognostic markers, as well as a tendency to higher mortality. Additionally, we identified clinical and laboratory predictors for HT, which may in a near future direct adapted-risk therapeutic strategies.

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CIRCULATING CELL-FREE DNA (CCFDNA) CONCENTRATIONS MEASURED BY CAPILLARY ELECTROPHORESIS: HIGH COST-EFFECTIVENESS IN DISCRIMINATING DLBCL PATIENTS AND ASSOCIATIONS WITH HIGH TUMOR BURDEN

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Introduction: DLBCL is a malignancy with marked clinical, biological and prognostic heterogeneity. Its different outcomes may be predicted by the identification of molecular biomarkers. Therefore, quantification of circulating cell-free DNA (ccfDNA) and circulating tumor DNA (ctDNA) have emerged as minimally invasive methods able to predict its prognosis. However, such measurements are based on the use of costly technologies and with restricted access in clinical practice. Here, we aimed to quantify ccfDNA in DLBCL using capillary electrophoresis (CEF), a low-cost and widely available methodology, in order to establish prognostic associations between its serum concentrations, clinical characteristics and outcomes. **Methods:** This prospective study involved 54 DLBCL patients, diagnosed at USP, from 2019 to 2020, and 23 healthy-controls. All cases were treated with 6-8 cycles of R-CHOP. Plasma samples were collected from DLBCL at three moments: at diagnosis, after the 4th CT cycle and at the end of treatment. ccfDNA was extracted using the QIAamp MinElute ccfDNA kit and quantified by CEF (Agilent 2100 Bioanalyzer). Comparison between two groups was performed using the Mann-Whitney test, and between three groups using the Kruskal-Wallis test. The cut-off for [ccfDNA] (1147 ng/ μ l) was determined by ROC methodology. Survival curves were constructed using the KM method and Log-Rank test was used to assess the relationship between variables and outcomes, and $p \leq 0.05$ was considered significant. **Results:** The median age at diagnosis was 57 years and 61.1% were female. Advanced-stage was observed in 59.3%, 29.6% had ECOG ≥ 2 , 52.6% had

IPI ≥ 3 , and 44.4% had bulky ≥ 10 cm. BM infiltration and involvement of ≥ 2 EN sites occurred in 5.6% and 16.8%, respectively. With a median follow-up of 32 months, the medians OS and EFS were 32 months (95% CI 28-35) and 31 months (95% CI 28-34), respectively. The ORR was 76% (95% CI 63.5-86). The overall mortality rate was 20.4% (95% CI 11.1-32.3). ccfDNA concentrations were significantly higher in DLBCL compared to controls ($p < 0.001$), however, it did not differ significantly between samples from DLBCL collected at the 3 different time points ($p = 0.602$). We observed positive associations between ccfDNA [] in diagnostic DLBCL samples and biomarkers of high tumor burden, including higher LDH levels ($p = 0.003$), bulky ($p = 0.011$), ECOG ≥ 2 ($p < 0.001$), and IPI ≥ 3 ($p = 0.069$). However, ccfDNA [] at diagnosis were not statistically associated with ORR ($p = 0.264$), nor with clinical outcomes, including OS (1-year OS: 92% for [ccfDNA] < 1147 ng/ μ l vs 76% for [ccfDNA] ≥ 1147 ng/ μ l, $p = 0.217$) and EFS (1-year EFS: 92% for [ccfDNA] < 1147 ng/ μ l vs 69% for [ccfDNA] ≥ 1147 ng/ μ l, $p = 0.270$). We believe that the short follow-up time and sample number restriction may have contributed to the lack of statistically significant association between ccfDNA [] and responses/outcomes observed. In this sense, cohort expansion and NGS techniques for ctDNA identification are ongoing to assess its potential impact as a prognostic biomarker in our cohort. **Conclusion:** We demonstrated that ccfDNA [] measured by CEF were able to accurately discriminate healthy individuals from DLBCL, highlighting its potential role as a minimally invasive diagnostic biomarker. Furthermore, ccfDNA [] at diagnosis in DLBCL patients were positively associated with markers of high tumor burden and adverse prognosis. Our results suggest that measurement of ccfDNA by CEF may be a highly cost-effective methodology to identify high-grade B-cell NHLs and recognize DLBCL subgroups with adverse clinical-prognostic characteristics.

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LESÃO RENAL AGUDA PÓS RENAL POR COMPRESSÃO DE LINFOMA DIFUSO DE GRANDES CÉLULAS B BULKY

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Introdução: A lesão renal aguda (LRA) é um quadro frequente dentro da clínica médica, havendo diversas etiologias, dentre elas a compressão extrínseca das vias urinárias. Esta compressão pode ser causada por tumores de origem local, como neoplasias ginecológicas, colorretal e, menos frequentemente, massas linfonodais de grandes dimensões. **Objetivo:** Relatar um caso de LRA pós renal devido compressão ureteral por linfoma difuso de grandes células B Bulky (LDGCB). **Relato de caso:** Paciente feminina, 45 anos, previamente hipertensa,