autologous stem-cell transplantation with median overall survival (mOS) exceeding 10 years. Real world evidence studies (RWE) compare clinical outcomes and population characteristics with the ones from clinical findings. **Objective:** We aimed to describe epidemiological characteristics, therapy and clinical outcomes of Brazilian patients with MCL diagnosticated between Jan 2010 until Dec 2021 and compare outcomes regarding the place of treatment (public vs private). This first report includes patients from 5 public and 1 private centers from 3 cities. **Patients and methods:** Patients with Cyclin D1 and/or SOX11 positivity were included. All the data is typed in an eCRF (Redcap by Einstein Hospital). Ki-67 values were retrieved from the pathological reports as percentages of positive MCL cells. A sample of patients from one public center was tested for P53 by immunohistochemistry (IHC) and results were scored as percentage of positive MCL cells. Statistical tests were done with SPSS 20.0 software. Survival curves were made by the Kaplan-Meier Method and compared with Log Rank test. **Results:** The study included 75 patients (56 males). Median age at diagnosis was 64 years (24y-96y). ECOG was 0 or 1 in 68 patients. MIPI risk-groups could be obtained in 63 patients and it was low in 18, intermediate in 23 and high in 22. The ki-67 was obtained in 43 patients and was ≤ 30% in 28, between 31%-49% in 11 and ≥ 50% in 12. The cytology was blastoid or pleomorphic in 8 of 24 patients. Treated in public institutions were 57 patients and 26 received intensive therapy among 56 patients with less than 70y at diagnosis. The mOS was 2.1 years (0.3-8.5) for the living patients, who were 31 at the last follow up. The analysis of mOS according to the MIPI was 2.5y for low, 2.7y for intermediary and 2.2y for high-score ($p = 0.98$). The Ki-67 (%) had an adverse prognosis at a cut off of 50% positive cells (3.2y vs 1.6y, $P = 0.01$). Patients with less than 70y had different mOS according to the type of treatment given (mOS for intensive therapy 3.3y vs 1.8y ($p = 0.04$) and there was no difference in mOS between patients treated at public vs private institutions ($p = 0.34$). We did p53 by IHC in 19 patients and there was a trend towards worse mOS for those with > 10% positive cells ($p = 0.08$). **Discussion:** We showed the first report from a MLC study collecting RWE from a population with low participation in MCL pivotal trials. The main finding is that the patients had worse overall survival than the ones reported in studies from developed countries. Unfortunately, morphology was not reported in a majority of patients and we should work on that. Our patients had a higher percentage of adverse prognostic factors such as a high MIPI and elevated KI-67. However, the previous was not prognostic and the latter was at a cut off of 50%. We will be looking closely on the factors that could explain this poor OS as the time to diagnosis/therapy and the availability of adequate therapy.

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ASSOCIAÇÃO ENTRE LINFADENOPATIA DERMOPÁTICA E LINFOMA DIFUSO DE GRANDES CÉLULAS B: UM RELATO DE CASO

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Objetivos: Relato de caso uma associação pouco comum entre Linfadenopatia Dermopática e Linfoma Difuso de Grandes Células B. **Material e métodos:** Trata-se de estudo retrospectivo, do tipo relato de caso. **Resultados:** Paciente do sexo masculino, 71 anos, encaminhado ao ambulatório de Hematologia para investigação de linfonodomegalias, predominantemente em região inguinal, visualizadas em ressonância magnética, em contexto de investigação de dor óssea. Não apresentava lesões de pele. Foi submetido a biópsia excisional de linfonodo inguinal, que revelou infiltrado histiocitário sem atipias e depósito de melanina no interior de macrófagos, com positividade para Ki67, CD20, CD3 e BCL2 à imunohistoquímica, achados típicos de linfadenopatia dermopática. Evoluiu com aumento importante e progressivo de linfonodomegalia inguinal além de surgimento de novas linfonodomegalias, o que não era compatível com linfadenopatia dermopática. Solicitada nova biópsia linfonodal, porém enquanto aguardava apresentou quadro de abdome agudo perfurativo por úlcera gástrica. Foi realizada biópsia desta lesão, cujos achados foram compatíveis com linfoma difuso de grandes células B, com positividade para CD20, BCL6, MUM1, BCL2. Foi iniciado tratamento com protocolo R-CHOP, com boa resposta clínica. **Discussão:** A linfadenopatia dermatopática é uma entidade distinta de hiperplasia linfoide benigna associada a uma variedade de doenças crônicas da pele, incluindo pênfigo, psoríase, eczema, neurodermatite e atrofia senil. Acredita-se que aconteça por uma resposta imune exagerada à estimulação antigênica persistente na pele. Há relatos menos frequentes de associação com outras doenças, inclusive neoplasias. Acomete principalmente linfonodos axilares e inguinais. Constitui um importante diagnóstico diferencial de linfonodomegalia. Seu diagnóstico é confirmado através de exame anatomopatológico do linfonodo, que mostra hiperplasia paracortical por células dendríticas, células de Langerhans, linfócitos e histiócitos contendo melanina. A imunohistoquímica revela positividade de CD68 e S100 nos histiócitos. No caso relatado, o achado de linfadenopatia dermopática chamava atenção para outra patologia de base, já que não havia correlação clínica, considerando rápido aumento de linfonodomegalia e ausência de lesões de pele. Posteriormente, foi confirmada a associação desta condição benigna a um linfoma difuso de grandes células B. Tal achado mostra a importância de realizar nova biópsia em caso de persistência de suspeição para doença linfoproliferativa, se necessário em sítios diferentes. **Conclusão:** O caso relatado mostra coexistência de linfadenopatia dermopática, de etiologia benigna, e linfoma difuso de grandes células B. Assim, a linfadenopatia dermopática pode representar um achado reacional de outras condições. Conclui-se que, caso haja evolução clínica diferente do esperado, deve-se seguir na busca pelo diagnóstico através de novas biópsias.

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