especialmente linfonodos. Esses tumores podem ser resultado de mutações, toxinas, infecções ou inflamação crônica. Atingem indivíduos com idade média de 67 anos, e incidência aproximada de 20/100.000 habitantes/ano. Em 2019, a pandemia por Coronavírus 2 ou coronavírus disease 19 (COVID-19) resultou na morte de mais de 6 milhões de pessoas em todo o mundo e impactou o cuidado de pacientes específicos, incluindo os imunossuprimidos, como os com LNH, que tem maior susceptibilidade a piores desfechos, quando acometidos pela COVID-19. Os aprendizados com a pandemia foram inúmeros e a vacinação, também trouxe novos desafios como a linfadenopatia persistente, que pode simular quadros de linfoma, inclusive com PET / CT positivo. Esse estudo visa relatar um caso de linfadenopatia persistente após a vacinação, simulando recidiva de LNH e revisar esta literatura. **Métodos:** Relato de caso, feita a partir da revisão de prontuário médico e com a permissão escrita do paciente. Revisão da literatura por busca não sistematizada nas plataformas PubMed e Scielo. **Resultados:** V.B, masculino, 24 anos, LNH difuso de grandes células B diagnosticado em agosto de 2016. Tratado com 6 ciclos de R-CHOP. Em remissão desde a última quimioterapia há 7 anos. Retornou para consulta de seguimento em junho/2021, com linfonodomegalia jugular posterior de 3 cm, tensa e indolor que surgiu 3 semanas antes, sem outros achados. Realizou novo PET/CT (julho 2021) e biópsia excisional do linfonodo (agosto de 2021). O PET/CT evidenciou linfonodomegalias cervicais nos níveis I, II, III e IV bilaterais e V à direita, com aumento de captação de FDG, Lugano 5. No entanto, o anatomopatológico e imuno-histoquímica do linfonodo evidenciaram hiperplasia linfoide reativa. Foi feita uma revisão de ambos os estudos que também resultou negativa para neoplasia. O paciente foi reavaliado em dezembro/2021, realizou novos PET/CT e biópsia, tendo achados semelhantes. O paciente recebeu 4 doses de vacina contra COVID-19: Coronavac (fevereiro e março/2021); Pfizer (dezembro/2021 e abril/2023). As sorologias para EBV e HIV eram negativas. Este paciente mantém acompanhamento e persiste com linfonodomegalia cervical, sem outros sintomas. **Discussão:** A linfadenopatia associada a infecção por COVID-19 com achados de hipercaptação ao PET/CT e linfadenopatia em cadeia mediastinal têm sido relatados. Porém, é mais comum após a vacinação. Ocorrem após a vacina de RNA mensageiro (14%) e vírus inativado (6,9%), especialmente após o reforço. Desses casos, o linfonodo hipercaptante costuma ser localizado em cadeia axilar, ipsilateral à aplicação, com resolução após cerca de 6 semanas. Existem poucos relatos de linfadenopatia cervical na literatura. Nesse caso, temos linfadenopatia persistente, em cadeia cervical posterior bilateral, com aumento de metabolismo ao PET/CT, simulando a recorrência de LNH, o que foi afastado por biópsia e imunohistoquímica em duas ocasiões. **Conclusão:** Linfadenopatia persistente relativa à vacinação em pacientes com história de neoplasias hematológicas em remissão é um possível fator de confusão no seguimento desses pacientes.

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## SÍNDROMES MIELODISPLÁSICAS

### TRANSCRIPTOMIC PUZZLE IN MYELODYSPLASTIC NEOPLASMS: UNRAVELING THE DISCORDANCE BETWEEN CD34+ STEM CELLS AND PRIMARY BONE MARROW CELLS

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**Introduction:** Differentially expressed genes (DEGs) biomarkers have the potential to aid in the diagnosis and monitoring of diseases, as well as determining the most effective treatments. However, due to the complex nature of Myelodysplastic neoplasm (MDS), it is challenging to assess the impact and variations of DEGs between CD34+ HSC (hematopoietic stem cells) and primary bone marrow cells (PBMC) in MDS pathogenesis. Consequently, this aspect remains largely unknown. **Objective:** The aim was to investigate the divergences in differential gene expression patterns between these two cell types as potential pathogenic biomarkers for MDS. **Materials and Methods:** To understand the transcriptome differential expression in MDS pathogenesis, we employed two groups: first, four datasets (GSE18366, GSE19429, GSE30195 and GSE58831) from CD34+ HSC samples were obtained to analyze expression profiles of 392 MDS patients and 44 healthy individuals; second, four other datasets (GSE41130, GSE97064, GSE107400 and GSE114869) from PBMC samples were obtained to analyze expression profiles of 700 MDS patients and 47 healthy individuals. All databases were based on Affymetrix GeneChip Human Transcriptome Array. We employed bioinformatic analysis of microarray to investigate significant abnormal differentially expressed genes (DEGs) in MDS and health individuals on every microarray dataset for both types of samples based on Transcriptome Analysis Console (TAC) Software algorithm. Heatmaps, Volcano and Scatter plots were performed in TAC software based in R algorithm. Gene Ontology (GO) enrichment and Kyoto Encyclopedia of Genes and Genomes (KEGG) pathways analysis were conducted to clarify the functional roles of DEGs. Enriched functional categories of gene–ontology associations were performed using the R software and the Database for Annotation, Visualization and Integrated Discovery (DAVID) database. **Results:** Our analysis initially revealed a disparity of 7117 expressed transcripts between PBMC (n = 40,165) and CD34+ HSC (n = 33,048). Furthermore, we identified 240 DEGs in CD34+ HSC samples and 2948 DEGs in PBMC samples. Among them, it was notable that there is no common pattern of gene expression between upregulated or downregulated transcripts in both samples. In this study, we conducted GO enrichment and KEGG pathway analyses to investigate the

functional roles of DEGs in Myelodysplastic Syndrome (MDS) pathobiology. We categorized the DEGs of CD34+ HSC and PBMC into functional categories using GO analysis. In CD34+ HSC, the most enriched pathways were signal transduction, negative regulation of transcription, and immune response. In PBMC, the immune response, signal transduction, and innate immune response pathways were significantly activated. We also performed KEGG pathway analysis for upregulated and downregulated DEGs in both CD34+ HSC and PBMC samples. In CD34+ HSC, the top pathways were involved in hematopoietic cell lineage, cell receptor signaling, and primary immunodeficiency. These pathways were also identified in PBMC, along with cancer and cell cycle pathways. **Discussion and Conclusion:** To summarize, our study uncovered disparities in DEGs between CD34+ HSC and PBMC cell types in MDS. However, we also observed a certain degree of similarity in the activated pathways of both cell samples based on Gene Ontology and KEGG pathways enrichment analyses. Of particular importance in our study was the role of the immune system and the activation of hematopoietic cell lineage pathways as the most activated in MDS, regardless of the cell type evaluated. These findings provide novel insights into DEGs biomarkers associated with MDS pathogenesis, holding clinical significance.

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## CASE REPORT: ADVANCED SYSTEMIC MASTOCYTOSIS ASSOCIATED WITH HEMATOLOGICAL NEOPLASIA

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**Objective:** Report a clinical case of Advanced Systemic Mastocytosis Associated with Hematological Neoplasia (MAS-ANH) under treatment with the AZAVIT-ABCDEF protocol, 7 days every 28 days (azacytidine associated with high doses of vitamins B1, C, and D, with erythropoietin and filgrastim). Brazil Platform CAAE: 53015421.0.0000.5463. After obtaining informed consent. **Methodology:** Assessment of patient's information from HSPE's computer-based patient records. **Clinical case:** A 71-year-old male with fatigue, diarrhea, pruritus, and extensive skin lesions in the groin, lymph node enlargement, and splenomegaly. Hb = 8.2 g/dL, platelets = 64,000/mm$^3$, and leukocytes = 11.38 thousand/mm$^3$ (1/5/54/0/0/37/3), Alkaline phosphatase = 239 U/L (30-120 U/L). Myelogram showed dysplastic changes, 4% blasts, and moderate mastocytosis. Bone marrow biopsy revealed 60% cellularity and mastocytosis. Immunohistochemistry: CD117 (+). Lymph node and skin biopsy showed infiltration by mast cells. Bone scintigraphy showed increased uptake in the skull, pelvis, spine, and femurs. Initially treated with alpha interferon, which resulted in increased cytopenias and worsening of constitutional symptoms. Decided to start zoledronic acid and AZAVIT-ABCDEF protocol, receiving 27 cycles every 28 days. Showed improvement in hemogram with sporadic blood component transfusions and reduction in splenomegaly. Pruritus and diarrhea improved significantly, and the patient gained weight. Triptase level still at 145 $\mu$g/mL (normal = 11 $\mu$g/mL) with slight improvement in skin lesions. Current blood count: Hb = 10.8 g/dL, platelets = 71,000/mm$^3$, and leukocytes = 9.280/mm$^3$ (1/5/59/1/0/14/20). **Discussion:** MAS-ANH is a rare entity associated with a mutation in the KIT D816V gene that encodes the receptor for stem cell factor (CD117). Additional mutations are commonly found, such as TET2 (29%), ASXL1 (17%), and CBL (11%). The main manifestations are hematological and gastrointestinal disturbances. Other symptoms include pruritus, urticaria, skin infiltration, bone pain, fatigue, fever, headache, and anaphylactic reactions. A prognostic index showed worse outcomes for cases with age > 60 years, thrombocytopenia, anemia, elevated alkaline phosphatase, and the presence of adverse mutations (ASXL1, RUNX1, and NRAS). Treatment options include KIT gene inhibitors (midostaurin, imatinib, and avapritinib), alpha interferon, and 2-CDA, with short-lived responses and the possibility of worsening cytopenias. Antihistamines and corticosteroids are used symptomatically. The good hematological response observed in this case probably occurred due to the presence of hematological clonal abnormalities resembling myelodysplastic neoplasia without mastocytosis. We believe that the protocol promotes differentiation by increasing the transcription of factors related to cell differentiation and improving aerobic metabolism. **Conclusion:** MAS-ANH is an aggressive pathology with a median survival of 20 months, even with the use of KIT gene loop inhibitors. At diagnosis, the patient presented with 4 out of 5 poor prognostic features and has already achieved a survival of 41 months. Further studies will be required to confirm the improvement in quality of life and survival with the AZAVIT-ABCDEF protocol.

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## PLATELET DYSFUNCTION IN A PATIENT WITH CHRONIC MYELOMONOCYTIC LEUKEMIA (CMML) AND RECURRENT SUBDURAL HEMATOMA.

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**Objectives:** To report a case of a patient diagnosed with CMML with recurrent subdural hematomas and platelet dysfunction. **Materials and methods:** The case report was based on data recorded in the electronic medical record and literature review. **Results:** A 62-year-old female diagnosed with CMML in February 2022 during investigation of persistent monocytosis associated with anemia and thrombocytopenia. Hemoglobin: 8.9 g/dL; White blood cell count: 11,860/mm$^3$ (69/0/0/9/20); Platelets: 45,000/mm$^3$. BCR-ABL and JAK2 tests were negative. Bone marrow examination revealed erythroid and granulocytic hypercellularity with dysplastic changes.