maneira mais minuciosa. Entender a linha de cuidado atual nos ajuda a avaliar com mais precisão os desfechos positivos e negativos, facilitando a implementação de ações mais efetivas, que contribuam para um melhor prognóstico do paciente.

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FLT3 VARIANTS IN PATIENTS WITH ACUTE MYELOID LEUKEMIA FROM AMAZONAS STATE, BRAZIL

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Background: Acute Myeloid Leukemia a cancer of the blood and bone marrow (BM), characterized by accumulation of leukemic blasts in the BM and peripheral blood. Among the variants identified in the AML, mostly occurs in the gene FMS-like tyrosine kinase 3 (*FLT3*), occurring approximately 10-30% of the cases. *FLT3* variants, as in-frame duplications of 3 to >400 base pairs, known as internal tandem duplications (ITDs) and tyrosine kinase domain (TKD) are more prevalent, 25% and 7-10%, respectively. **Objective:** We investigated genetic variants in the *FLT3* in patients with AML, also evaluated possible association of the *FLT3* variants with clinical and laboratory data. **Methods:** The study population was composed of 18 AML patients. The nucleotide sequencing was performed in bone marrow samples, sequencing directed for ITD and TKD region (exon 9-24). **Results:** The patients included, ten (56%) are females and eight (44%) males, being twelve (67%) primary AML patients, three (16.5%) secondary AML and three patients (16.5%) with relapsed. The missense variants in the *FLT3* were identified in four patients: Y416N (exon 10), V557I (exon 13), A680V (exon 16), Y702S (exon 17) and *FLT3-ITD* (exon 14). The $FLT3^+$ variants were observed in two patients (50%) with secondary AML, one patient (25%) with secondary AML to myelodysplasia and one patient (25%) with relapsed. According to hematological data, the percentage of blasts was higher in $FLT3^+$ patients when compared to $FLT3^-$ patients, 40.25% (±31.35) and 35.23% (±30.5), respectively. The white blood cell count was high in $FLT3^-$ patients when compared to $FLT3^+$ patients [14,500 mm^3 (2,300-61,390) vs 10,225 mm^3 (5,210-10,808)], however low red blood cell count was observed in $FLT3^+$ patients 2.6 mm^3 (±0.77), 2.96 mm^3 (±0.39) in $FLT3^-$ patients. Moreover, hemoglobin was higher in $FLT3^+$ patients when compared to $FLT3^-$ patients [9,6 g/dL (5,9 -10,8) vs 8,3 g/dL (7,0- 10,6)], as well, platelet count in $FLT3^+$ patients [48,500 mm^3 (25,500-65,500) vs 35,000 mm^3 (15,000-51,000)]. **Discussion:** Previous studies have reported that the frequency *FLT3* variants occurs in approximately 10-30% of the AML cases, in this study was observed in 22% of the cases. Different studies highlight the *FLT3-ITD* as variant more prevalent of the *FLT3*, however, here related the TKD and extracellular domain variants as more prevalent with 50% and 30%, respectively. These found may be due to the features of the study population in our region from Amazonas. **Conclusion:** Variants different are identified in the *FLT3*, we highlight the importance to investigate other variants located in the *FLT3* exome in AML patients, as well as, to evaluate possible association with change hematological, as platelet and red blood cell count.

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EXPLORING THE EFFICACY OF AZACITIDINE AND VENETOCLAX COMBINATION IN TREATING ACUTE MYELOID LEUKEMIA IN CASES OF RELAPSE FOLLOWING AZACITIDINE TREATMENT AND HIGH-DOSE VITAMINS THERAPY

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Objective: Report of 4 cases monitored at the do Servidor Publico Estadual Hospital (IAMSPE), SP. Patients considered unfit for 3+7 regimen received AZAVIT-ABCEF (azacytidine, filgrastim, erythropoietin combined with high doses of vitamins B1, C, and D3). In cases of relapse, they were treated with azacytidine 75 mg/m^2 and venetoclax 400 mg for 7 days every 28 days (aza + veneto). **Methodology:** Data collection from the IAMSPE digital system. **Results: Patient 1:** Female, 77 years old, AML diagnosed in Oct/21. Hemogram: blasts=36%, Bone Marrow: blasts = 78%, Immunophenotype (IFT): CD13, CD15, CD33, CD34, CD71, CD117, CD123, HLA-DR. FISH: KMT2 gene trisomy in 45/200 metaphases; Karyotype: 47, XX, +11[3] /46, XX[17]. Received AZAVIT-ABCDEF for 15 months without transfusion need until worsened hemogram in Jan/23; Bone Marrow: blasts = 56%. Started aza + veneto in Feb/23, after 1 cycle, bone marrow: 3% immature cells and monocytosis of 13%. Sustained pancytopenia requiring blood component support for 55 days. Cycle 2 delayed, currently on 6th cycle, Hemogram JUL/2023: Hb = 13.8 g/dL, WBC = 6030/mm^3 (62.1,70.0,2.32.4,1), Platelets = 317000. **Patient 2:** Female, 71 years old, AML diagnosed in Jan/22. Hemogram: monocytosis = 55%, Bone Marrow: blasts = 94%, IFT: CD13, CD14, CD15, CD33 CD36, CD56, CD64, HLA-DR, MPO. FISH and Karyotype: normal. Received AZAVIT-ABCDEF for 11 months without transfusion need until worsened hemogram monocytes = 58%, Bone Marrow: blasts = 67%. Started aza + veneto in Jan/23, currently on 7th cycle. Current Hemogram: Hb = 14.5 g/dL. WBC = 3000 (2/42/1/0/39/16), Platelets = 204. **Patient 3:** Female, 69 years old, AML diagnosed in May/22. Hemogram: bicytopenia, Bone Marrow: blasts = 79%, IFT: CD4, partial CD11b, partial CD13, CD15, CD33, CD36, CD38, CD64,

CD56, CD123, HLA-DR, MPO. FISH and Karyotype: normal. Received AZAVIT-ABCDEF for 8 months without transfusion need until worsened hemogram in Jan/23; Bone Marrow: blasts = 35%. Started aza + veneto in Feb/23, after 1 cycle, patient developed pancytopenia and marrow showed maturation arrest. Patient had cycles postponed, completed 3 cycles with sporadic blood component need. JUL/23: Hb = 7.6 g/dL. WBC = 4020 (1/1/3/38/0/0/52/5), Platelets = 34000. **Patient 4:** Female, 58 years old, AML diagnosed in Oct/22. Hemogram: blasts = 6%, Bone Marrow: blasts = 21%, IFT: CD13, CD33, CD34, CD38, CD117, CD123, HLA-DR, MPO. FISH and Karyotype: normal. Received AZAVIT-ABCDEF for 8 months without transfusion need until worsened hemogram in Jun/23; Bone Marrow: blasts = 35%. Started aza + veneto in Jun/23, after 1 cycle, achieved remission; in the second cycle, patient developed prolonged pancytopenia and awaits marrow recovery. **Discussion:** AZAVIT-ABCDEF protocol potentially enhances mitochondrial metabolism and discourages anaerobic metabolism, inducing leukemic cells to enter the Krebs cycle and promoting differentiation. Using venetoclax during relapse possibly exploited leukemic cells' sensitivity to mitochondrial blockade with anti-BCL2, explaining the positive response in all 4 patients. **Conclusion:** The patients demonstrated an improved quality of life with increased overall survival. In this pathology where the diagnosis is practically a death sentence for individuals in this age group or with comorbidities, the results are promising.

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AVALIAÇÃO DOS NÍVEIS DE PODOPLANINA SOLÚVEL EM PACIENTES COM LEUCEMIA PROMIELOCÍTICA AGUDA E SUA ASSOCIAÇÃO COM A ATIVAÇÃO PLAQUETÁRIA

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Introdução: a coagulopatia da leucemia promielocítica aguda (LPA) é responsável por uma taxa extremamente alta de sangramento e morte precoce em um grupo de pacientes que apresenta um prognóstico de longo prazo melhor em comparação com outras formas de leucemia mieloide aguda (LMA). Recentemente, a expressão de podoplanina (PDPN), uma proteína que medeia a ativação plaquetária em contextos inflamatórios, por células leucêmicas foi descrita como relevante nessa coagulopatia. **Objetivo:** Explorar se os níveis circulantes de PDPN podem ser usados como um biomarcador de LPA e se seus níveis estão associados a marcadores clínicos ou laboratoriais de ativação plaquetária e de coagulação em LPA. **Materiais e métodos:** as amostras foram obtidas de pacientes consecutivos com LPA no momento do diagnóstico no Hospital das Clínicas da Unicamp. Amostras do biobanco de 35 pacientes com outras LMAs pareadas por idade e sexo foram usadas em análises comparativas. Os dados clínicos e laboratoriais foram extraídos dos prontuários. Os níveis circulantes de PDPN foram medidos no plasma (EDTA) usando um kit Elisa comercial. Biomarcadores de ativação plaquetária e de coagulação foram medidos usando ensaios imunológicos multiplex. O estudo foi aprovado pelo comitê de ética e todos os participantes forneceram consentimento informado por escrito. **Resultados:** os pacientes com LPA estão classificados em grupos de riscos, sendo eles baixo (3 pacientes), intermediário (17 pacientes) e alto (15 pacientes). A frequência de sangramentos, sangramento de SNC e trombose foi respectivamente de 31%, 11% e 8,5%. O fibrinogênio foi menor nos pacientes com LPA quando comparado as outras formas de LMA (115,4 (52,2-376,0) vs 383,2 (147,6-638,2) mg/dL, U = 30,00; $p < 0,0001$). O teste de Mann-Whitney mostrou que os níveis de PDPN foram significativamente maiores nos pacientes com LPA em comparação com outras formas de LMA (16,4 (0,5-120,5) vs 2,4 (0,37-238,9) ng/mL, U = 399,00; p = 0,01). Os níveis de P-selectina (7.194 (2.213-30.650) vs 11.292 (2.792-235.307) pg/mL, U = 357,00; p = 0,002) e CD40 ligante (100,4 (13,7-559,8) vs 196,6 (89,6-575,9) pg/mL, U = 276,00; p = 0,002) foram menores nos pacientes com LPA quando comparados as outras formas de LMA. Dentre os parâmetros testados, a PDPN correlacionou-se com o biomarcador clássico de ativação plaquetária (CD40L) em pacientes com LPA ($R_S = 0.603$; p = 0,0004). No entanto, os níveis de PDPN não foram associados ao desenvolvimento de sangramento ou trombose entre os pacientes com LPA nesta coorte. **Discussão e conclusão:** os níveis circulantes de PDPN são mais elevados em LPA e correlacionam-se com evidências laboratoriais de ativação plaquetária. Estudos adicionais são necessários para confirmar esses achados e para explorar ainda mais a associação de PDPN com a apresentação clínica de coagulopatia na LPA.

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OUTCOMES IN PATIENTS WITH B-CELL PRECURSOR ACUTE LYMPHOBLASTIC LEUKEMIA RECEIVING INOTUZUMAB OZOGAMICIN AND PROCEEDING TO HEMATOPOIETIC STEM CELL TRANSPLANTATION

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