infecção fetal, crise aplástica e, em imunocomprometidos, aplasia pura de série vermelha. **Relato de caso:** Paciente do sexo masculino, 27 anos, diagnosticado em outubro de 2023 com leucemia mieloide aguda de risco citogenético favorável (CBF-MYH11,FLT3-ITD detectado). Recebeu tratamento de indução com esquema composto por citarabina e daunorrubicina ("7+3") e obteve remissão completa. À admissão para primeiro ciclo de consolidação, exames laboratoriais evidenciaram anemia grave microcítica e hipocrômica (hb 6,1 g/dL,VCM 78), hipoproliferativa (reticulócitos de 0,03%), sem ferropenia e sem sinais de hemólise apesar de Coombs direto. Encontrava-se afebril, sem rash cutâneo. Mielograma demonstrou hipoplasia isolada da série eritrocítica, com índice granulocítico/ eritroidede 96. Não havia aumento de blastos. Biópsia de medula óssea com celularidade normal para idade, com série eritrocítica extremamente hipocelular. Em ambos, observava-se proeritroblastos (e-caderina positiva e glicoforina negativa) gigantes com inclusões nucleares proeminentes e vacuolizações citoplasmáticas, característicos da infecção peloparvovírus B19. Foi realizada investigação sorológica, com IgG eIgM, inicialmente negativos, mas com soroconversão em nova amostra após 5 semanas. Paciente recebeu tratamento de suporte com transfusão de hemácias. Houve lenta recuperação da série eritrocítica, o que postergou o início da fase de consolidação em um mês. Foi submetido a dois ciclos de consolidação e completa, no momento, oito meses em remissão. **Discussão:** apesar de mais frequentemente associado ao eritema infeccioso em crianças e adolescentes, o parvovírus B19, em hospedeiros imunocomprometidos, incluindo portadores de neoplasias hematológicas em tratamento, está associado a manifestações clínicas distintas, que podem resultar de infecção primária ou de reativação viral. Tais manifestações podem incluir rash cutâneo na fase precoce, citopenias na fase tardia e falência orgânica. O quadro infeccioso pode se confundir com recidiva da doença hematológica ou toxicidade ao tratamento, sendo necessário alto índice de suspeição para o diagnóstico. **Conclusão:** A infecção por parvovírus B19 pode causar aplasia pura de série vermelha em pacientes imunocomprometidos, como no caso relatado, onde houve, inclusive, atraso no tratamento da LMA. É necessário alta suspeição para seu diagnóstico, tanto suspeita clínica, quanto morfológica. É crucial, também, enfatizar práticas corretas de higiene e controle de infecção a fim de prevenir transmissões virais.

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IMPLICATIONS AND ASSOCIATION OF TP53BP2, BCL2L11, SOD2, CAT GENES IN HEMATOLOGICAL DISEASES

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Objectives: Differential diagnosis of hematologic diseases is a huge challenge and advances in genetic studies have made it possible to unravel the many gene interactions in these diseases. In this study, we performed a literature search on the roles of the TP53BP2, BCL2L11, SOD2 and CAT genes in cellular regulatory processes and mechanisms and describe the implications reported in the literature in association with hematologic diseases. **Materials and methods:** This is an integrative review of the literature from the last 10 years (2015-2024), carried out on the PubMed and Google Scholar platforms, using the following keywords: "hematological diseases", "implications", "gene expression", "polymorphisms", also using NCBI GENE. Of the total of 87 articles found, only 22 aimed at and covered the clinical focus of the hypothesis of this review. **Results:** Several genetic alterations and mutations were described in these genes, with emphasis on individualized SNPs as factors predominantly involved in the development and progression of hematologic malignancies. Most of these highlighted the suppression and silencing of the TP53BP2 and BCL2L11 genes, which play a crucial role in cell death. Oncogenic splice variants (ASPP2k) stood out influencing gene expression, downregulation and interactions with nuclear viral antigens, all of which converge to the deregulation of the apoptosis mechanism, implying susceptibility, proliferation, invasiveness, resistance to treatment and a marker of unfavorable prognosis. In addition, polymorphisms in SOD2 (rs4880) directly contribute to vascular and cerebral damage and complications in sickle cell anemia patients; in addition to being associated with hepatotoxicity in ALL patients; Another SOD2 SNP (rs8031) has been implicated in many studies of patients with refractory AML due to increased resistance to treatment. The SNP rs1001179 in the CAT gene is involved in the dysregulation of antioxidant mechanisms, being correlated with a more aggressive course in CLL patients. **Discussion:** Altered expression of genes involved in apoptotic regulation is frequently associated with several types of cancer and notably contributes to cell proliferation and resistance to chemotherapy, due to impaired apoptosis specifically in multiple myeloma where reduced gene expression has been observed. The diversity and complexity of hematologic malignancies demonstrate the need for an individualized understanding of the underlying causes. Altered gene expression, downregulation, and polymorphisms of many genes suggest the possibility of delineating populations susceptible to the disease to propose an early approach, in addition to suggesting a possible prediction of severity and response to treatment. **Conclusion:** Discoveries in these genes may expand early diagnosis, predict disease course, and individualized adaptations in therapy. In this review, we explore

the importance of genes that are critical in the pathogenesis, progression, and are potentially essential in the treatment of hematologic malignancies. This study highlights the importance of genetic understanding in the study of large populations, and we believe that the integration of these genetic and molecular discoveries presupposes the transformation of the management of these diseases.

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PLASMINOGEN ACTIVATING RECEPTOR UROKINASE (U-PAR): INTERACTION AND IMPLICATIONS IN TUMOR MICROENVIRONMENT IN LEUKEMIAS

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Objectives: The urokinase-like plasminogen activator receptor (uPAR) interacts with its ligand (uPA) to convert plasminogen into plasmin, which degrades components of the extracellular matrix. Plasminogen activation can influence many normal and pathological processes through interactions that culminate in cell activation, adhesion, migration and extravasation. This study aimed to describe the implications of uPAR-mediated plasminogen activation in leukemias, exploring its interaction with the tumor microenvironment. **Materials and methods:** This is an integrative review following the Preferred Reporting Items for Systematic Reviews guidelines for reports, searching for published data available on the descriptors “uPAR”, “leukemia” and “genetic expression”. The research was carried out on the MEDLINE (PubMed), Science Direct, Google Scholar and Scielo platforms, covering the last 10 years (2015-2024) with only 23 articles meeting the criteria for approaching the proposed theme. **Results:** Studies have shown high levels of suPAR and positive surface expression of uPAR associated with decreased chemosensitivity in patients with M0-M5 AML, where blasts expressing the surface receptor have been observed. The increase in uPAR levels was related to hyperfibrinolysis in patients with acute promyelocytic leukemia (APL) and associated with increased invasive proteolytic activity, interaction of tumor cells with the immunosuppressive microenvironment, causing immune evasion and dysfunction of effector cells. Similar results were demonstrated in patients with AML, ALL and biphenotypic leukemia, where high uPAR expression was associated with the invasive capacity of leukemia cells, and involved in the interaction of tumor cells with a suppressive microenvironment, which may influence the lymphocyte-mediated immune response. In addition to observations on altered expression, transcription variants were identified that can act as ceRNAs (uPAR Δ5/ Δ6/ uPAR Δ6/7) in myeloid lineage cells, demonstrating higher levels in monoblasts. **Discussion:** The high expression of the receptor in leukemic blasts of AML M0-M5 evidences its role in the invasion and metastasis of malignant cells. The ability of the receptor to influence the immune response has been shown to be an indicator of poor prognosis and a potential marker of risk stratification. The direct interaction of stromal and leukemic cells denotes the formation of a competitive niche that can favor malignant cells. In addition, the identified variants suggest a negative modulation in the cell-matrix interaction, favoring the proliferation and progression of leukemia. In short, if there is no conversion of plasminogen into plasmin, which is also involved in angiogenesis, it leads to a decrease in nutrients and O2 in leukemia cells. **Conclusion:** The studies pointed to the contribution of the receptor in tumor progression, immune evasion and resistance to treatment, suggesting that the receptor is a target in the therapeutic potential, that is, the key molecule to limit the interactions of the plasminogen activation system, in order to suppress tumorigenesis. The recent development of anti-urokinase receptor antibodies has shown promise in blocking the interactions of the receptor and its ligand, thereby inhibiting the proteolytic cascade. In addition, the recipient can also improve the effectiveness of hematopoietic stem cell transplants.

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RECONSTITUIÇÃO DO SISTEMA IMUNOLÓGICO HUMANO EM MODELO MURINO IMUNOSSUPRIMIDO

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Objetivos: Padronizar o processo de humanização do sistema imune de camundongos imunodeficientes da linhagem NSG, utilizando células-tronco hematopoéticas CD34^{+}. **Materiais e métodos:** Amostras de sangue de cordão umbilical (UCB) humano foram obtidas do biobanco do Hospital da Mulher Prof. Dr. José Aristodemo Pinotti (CAISM/UNICAMP) e criopreservadas em nitrogênio líquido. Para a obtenção de