cases were pediatric (71%), with a median age of 8 years-old, and low white blood cell count at diagnosis (64%). *CDKN2A/B* del (47%) and *JAK2* del (17%) were concomitantly observed with PAX5alt. On the other hand, in *PAX5* wild-type patients, CDKN2A/Bdel (26%) and *IKZF1* del (20%) were the most prevalent. Curiously, the *IGH* -rearrangements were commonly found among *PAX5* alt cases (23%). *PAX5* P80R were not found in any cases. Considering that the genetic alterations knowledge is essential for the treatment decisions, a better understanding of *PAX5* alterations in B-other ALL is crucial for precision medicine. Besides, our findings suggest that the *IGH* gene may be a frequent partner of *PAX5* fusions, though it requires further investigation.

https://doi.org/10.1016/j.htct.2024.09.690

TP53 GENE EXPRESSION IS INCREASED AND CORRELATES WITH RHOC IN PATIENTS WITH ACUTE MYELOID LEUKEMIA

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Background: Mutations in the tumor suppressor gene *TP53* are found in approximately 10% of acute myeloid leukemia (AML) cases and are associated with a worse prognosis. Deregulation of the p53 signaling pathway occurs by other mechanisms in additional AML patients. Therefore, the investigation of new genes associated with p53 pathway is important to understand the physiopathology of *TP53* -mutated and *TP53* -wild type AML. In this context, RHO GTPases have been shown to participate in p53 signaling, but this relation has not been explored in AML. **Objectives:** In this study, we aimed to evaluate the expression of *TP53* and its regulators (*MDM2* and *CDKN2A)* in patients with *de novo* AML, and AML with myelodysplasia related changes (AML-MRC). We also investigated the correlation among *TP53*, *MDM2*, *CDKN2A, RHOA, RHOB, and RHOC* gene expressions. **Material and methods:** Gene expression was evaluated by quantitative PCR in bone marrow samples from healthy donors (n = 11) and patients with AML (n = 33), and AML-MRC (n = 15). This study was approved by the Institutional Ethics Committees. Groups were compared with the Mann-Whitney test. Correlations were performed with Spearman test. **Results:** *TP53* expression was increased in both *de novo* AML (p = 0.0022) and AML-MRC patients (P = 0.0008) compared to healthy donors. A decrease in *MDM2* gene expression was observed in *de novo* AML (P = 0.0101) and AML-MRC (p = 0.0422). Confirming our previous results, *RHOC* expression was also increased in both groups of AML patients (p = 0.0025 and p = 0.0129), whereas *RHOA* expression was decreased in AML-MRC patients (p = 0.0374). *CDKN2A* and *RHOB* expressions did not significantly differ among the groups. Additionally, *TP53* expression positively correlated with *RHOA* (r = 0.2949, p = 0.0338) and *RHOC* (r = 0.3373, p = 0.0145). **Conclusions:** *TP53* and *RHOC* gene expressions are increased and positively correlated in *de novo* AML and AML-MRC. The increase in *TP53* expression may be a compensatory mechanism to suppress disease progression. Moreover, a complex feedback mechanism between p53 and MDM2 could be responsible for the low expression of *MDM2*. Correlation between *TP53* and *RHOC* expression suggest a possible relation between these signaling pathways in AML. **Funding:** Funded by FAPESP.

https://doi.org/10.1016/j.htct.2024.09.691

LEUCEMIA MIELOMONOCÍTICA AGUDA EM PACIENTE COM MICRODELEÇÃO 7Q31 NÃO SINDRÔMICA E DOENÇA DE GRAVES: RELATO DE CASO

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Introdução: Leucemia mieloide aguda é a segunda forma mais comum de leucemias em adultos. Possui como fatores de risco radiação ionizante, tabagismo, quimioterapia, produtos químicos, doenças mieloproliferativas, neoplasia mielodisplásica e mutações cromossômicas. Doenças autoimunes também elevam o risco. A leucemia mielomonocítica aguda corresponde a 15-20% das leucemias mieloides agudas. **Relato de caso:** Mulher de 20 anos admitida por síndrome anêmica, gengivorragia e metrorragia há 02 semanas. Portadora de doença de Graves há 02 anos, em uso de Metimazol. Ao exame físico, presença de mucosas hipocoradas, proptose à esquerda com sinal de lid-lag, hiperplasia gengival, adenomegalias cervical, axilar e inguinal, hepatoesplenomegalia e equimoses em membros. Exames laboratoriais evidenciaram anemia grave normocítica normocrômica, leucocitose com 16% de células mononucleares atípicas apresentando características blásticas, além de plaquetopenia e hiperbilirrubinemia. Mielograma mostrando células com nucléolos evidentes, cromatina frouxa e alta relação núcleo-citoplasmática. Imunofenotipagem de medula óssea quantificado 37,70% de células imaturas da linhagem mieloide, apresentando fenótipo anômalo (hiperexpressão de CD13 e expressão parcial de CD1 1be CD117) além de 35,38% de células monociticas apresentando fenótipo maduro, confirmando-se o diagnóstico de leucemia mielomonocítica aguda. Análise do liquor também identificou 2,55% de mieloblasto anômalos. O cariótipo de medula óssea revelou deleção de cromossomo 7 em 20 metáfases, classificando-a como alto risco. Paciente foi transferida para centro de referência em onco-hematologia do estado da Bahia. Realizou indução quimioterápica com protocolo 7+3, atingindo remissão morfológica completa. Contudo, após dois ciclos de consolidação com citarabina em altas doses, manteve doença residual mínima (DRM) detectável por citometria de fluxo. Realizada quimioterapia intratecal com