detection of single nucleotide polymorphisms (SNPs), which recognize, in advance, patients who will develop hypersensitivity to ASNase, would help to optimize treatment. To describe pharmacogenetic variations influencing hypersensitivity to PEG-ASNase and their correlation with our patient population. **Material and methods:** We conducted a prospective multicenter study involving ALL patients under 18 years old receiving PEG-ASNase. Genotyping was carried out for 6 SNPs, which are more frequently related to hypersensitivity in other populations, including GRIA1 rs4958351, NFACT2 rs6021191, MYBBP1A rs3809849, CNOT3 rs73062673, GR1A1 rs6890057 and HLA-DQB1 rs1694129. The tests were performed using custom TaqMan® SNP genotyping assays. **Results:** A total of 305 patients were included, with 10.8% experiencing clinical allergic reactions and (14.1%) patients with inactivation (ASNase activity < 0.1 IU/mL). Genotypic distribution and Hardy-Weinberg equilibrium (HWE) for studied SNPs were as follows: GRIA1 rs4958351 (GG 59.1%, GA 32.6%, AA 8.3%, HWE = 0.06); NFACT2 rs6021191 (AA 89.8%, AT 9.8%, TT 4%, HWE = 0.74); MYBBP1A rs3809849 (GG 60.7%, GC 35.3%, CC 4%, HWE = 0.55); CNOT3 rs73062673 (TT 69.8%, TC 26.4%, CC 3.7%, HWE = 0.34); GR1A1 rs6890057 (CC 70.6%, CT 25.5%, TT 3.9%, HWE = 0.22); HLA-DQB1 rs1694129 (GG 83%, GT 16.5%, TT 4%, HWE = 0.55). No correlation was found between clinical allergic reactions and inactivation with SNPs ($p > 0.01$). **Discussion:** Our analysis characterized the pharmacogenetic polymorphisms in our population, reflecting diversity similar to the general population. Despite the lack of correlation between the studied SNPs and allergic reactions observed in our sample, we encourage further exploration to identify additional SNPs associated with hypersensitivity. **Conclusion:** These findings lay the groundwork for future research to uncover new genetic predictors of hypersensitivity, which could contribute to the development of personalized treatment approaches and the prevention of allergic reactions.

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CLINICAL AND MOLECULAR IMPACT OF RS3824662 POLYMORPHISM IN GATA3 IN THE DEVELOPMENT OF ACUTE LYMPHOBLASTIC LEUKEMIA IN ADOLESCENTS AND YOUNG ADULTS

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Introduction and objectives: The rs3824662 polymorphism in *GATA3* is a risk variant for the development of B-cell precursor acute lymphoblastic leukemia (B-ALL), particularly *BCR::ABL1-like* ALL, a subtype of B-ALL defined by a gene expression profile similar to the observed in *BCR::ABL1* positive patients. The predominance of high-risk alterations jointly with the absence of specific treatment protocols for adolescents and young adults (AYA) has negatively impacted the prognosis of this age subgroup. Besides that, studies evaluating the *GATA3* SNP in AYA are rarely observed. Given this scenario, this study aims to evaluate the clinical and molecular impact of the SNP rs3824662 on the development of B-ALL in AYA. **Material and methods:** 76 samples from AYA patients (15 to 39 years old) diagnosed with B-ALL according to the World Health Organization criteria were included between 2018 and 2023. This project was approved by the INCA Research Ethics Committee (#87793418.0.0000.5274). The samples were obtained from different cancer treatment centers in Brazil. DNA was extracted using the DNA MiniBlood kit (Qiagen) following the standard protocol. The rs3824662 polymorphism was identified using the TaqMan allelic discrimination assay (C_27522049_10, Life Technologies). The observed genotypes were validated by direct sequencing using the ABI 3500 Genetic Analyzer (Life Technologies). The *BCR::ABL1-like* subtype was characterized by the presence of rearrangements in *PDGFRB, CRLF2, JAK2, ABL1, ABL2, IGH*, and/or *CSF1R* evaluated by FISH. *GATA3* SNP was correlated with previously characterized clinical and molecular variables, including primary alterations detected by qPCR and additional alterations identified by the SALSA MLPA P335-C1 kit (MRC Holland). **Results:** We successfully analyzed 76 patients, displaying a median age of 19.5 years-old and the majority had a low white blood cell (WBC) count (77.0%). Regarding molecular subgroups, most of the patients did not present recurrent alterations and were characterized as B-other (35.9%). Heterozygote and mutant genotypes of rs3824662 SNP were observed in 39.7% and 8.2% of patients, respectively. The mutant allele A rarely occurred in concomitance with *BCR::ABL1* or *TCF3::PBX1*, and was particularly overrepresented in B-other patients and *BCR::ABL1-like* subtypes ($P < 0.05$). Additionally, it was more common in patients with low WBC count (86.1%). Curiously, patients with the mutant genotype had a very low frequency of deletions in *IKZF1, EBF1, JAK2, CDKN2A/B, PAX5, ETV6, BTG1, RB1*, and deletions in the PAR1 region were not observed ($P < 0.05$). **Discussion and conclusion:** Our findings are consistent with previous studies in AYA B-ALL, corroborating that rs3824662 SNP is a significant factor for the development of *BCR::ABL1-like* B-ALL. Furthermore, there is an underrepresentation of this SNP in concomitance with deletions in genes involved in differentiation, proliferation, and cell cycle that are frequently affected in B-ALL. These findings suggest that this SNP can directly impact the development and progression of leukemogenesis, and could be incorporated in the diagnostic routine of B-ALL patients. **Support:** FAPERJ, CNPq, Ministério da Saúde, Swiss Bridge Foundation.

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ANÁLISE DO PERFIL SOCIODEMOGRÁFICO DOS PACIENTES COM DIAGNÓSTICO DE DOENÇAS ONCO-HEMATOLÓGICAS, ATENDIDOS EM UM CENTRO DE REFERÊNCIA DE UM MUNICÍPIO DO SUL DE MINAS GERAIS.

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