with unexplained Rh antibodies. All patients had complete serological and molecular analyses. A lookback on the donor units transfused to those patients was performed and donors suspected of having Rh variants were recruited for further analysis. Laboratory and clinical findings were used to evaluate the clinical significance of the alloantibodies produced. **Results:** Unexpected Rh antibodies found in these patients were not linked to expression of partial Rh phenotypes according to serological and molecular analyses. Anti-D was found in 2 patients, anti-C was found in one patient, anti-c was found in one patient and anti-e was found in 3 patients carrying conventional D, C, c and e antigens respectively. Serological and molecular analyses on donors'samples revealed that 6 donors whose RBCs were transfused to these patients carried partial Rh antigens. Only one anti-e in a patient with β-thalassemia was autoreactive and could not be explained by RH diversity in his donors. Laboratory and clinical evidences of a delayed hemolytic transfusion or decreased survival of transfused RBCs were associated with 3 of 7 Rh antibodies at first detection. **Discussion:** Our study provides evidence that patients exposed to RBC units from donors with Rh variants may develop antibodies and some of these may be of clinical significance.

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SYSTEMATIC RHD GENOTYPING IN BRAZILIANS REVEALS A HIGH FREQUENCY OF PARTIAL D IN CHRONICALLY TRANSFUSED PATIENTS SEROLOGICALLY TYPED AS WEAK D

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Background: RHD molecular analysis has been used to differentiate weak D and partial D. This discrimination can be of clinical importance because carriers of partial D antigen may develop anti-D when transfused with D-positive red blood cell units what is not commonly observed in the weak D phenotype, with rare exceptions. The aim of this study was to determine the type of D variants among Brazilian patients requiring chronic transfusions serologically typed as weak D. **Methods:** Samples from 87 patients (53 with sickle cell disease, 10 with thalassemia and 24 with myelodysplastic syndrome), typed as weak D by manual tube indirect antiglobulin test or gel test were first RHD genotyped by using the RHD BeadChip Kit (BioArray, Immucor). Sanger sequencing was performed when necessary. **Results:** RHD molecular analysis revealed 32 (36.8%) variant RHD alleles encoding weak D phenotypes and 55 (63.2%) alleles encoding partial D antigens. RHD variant alleles were present in the homozygous state or as a single RHD allele, one variant RHD allele associated with the RHD Y allele, or two different variant RHD alleles in compound heterozygosity with each other in 67 patients, 6 patients and 14 patients respectively. The most common RHD alleles predicting partial D were RHD*DAR1.2, RHD*DAR3.1, RHD*DAU4, RHD*DAU*6, RHD*DVI, RHD*DVII, RHD*DMH while the most common RHD alleles predicting weak D were RHD*weak D type 1, RHD*weak D type 3, RHD*weak D type 4.0 and RHD*weak D type 38. Alloanti-D was found in 9 (16.4%) cases with variant RHD alleles (specifically RHD*DAR1.2, RHD*DAR3.1, RHD*DVI and RHD*DVII) predicting a partial D. **Conclusions:** The frequency of partial D was higher than weak D in Brazilian patients serologically typed as weak D, showing the importance to differentiate weak D and partial D in chronically transfused patients to establish a transfusion policy recommendation. Systematic RHD molecular analysis in patients receiving red blood cell transfusions provides relevant information of variant RHD alleles improving transfusion therapy and preventing alloimmunization. It also helps to avoid wasting of D- red blood cell units because carriers with the most common weak D types may safely receive D+ units.

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TÉCNICA DE BIOLOGIA MOLECULAR COMO ALTERNATIVA EM FENOTIPAGENS INCONCLUSIVAS

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Objetivos: O objetivo desse trabalho foi demonstrar a contribuição da técnica de genotipagem eritrocitária na prática transfusional. **Material e métodos:** Foram genotipadas 18 amostras de pacientes com fenotipagem inconclusiva ou incompleta. A extração do DNA das amostras foi realizada utilizando-se o conjunto "*iopur Kit Extração Mini Spin Plus*" (Mobius Life Sciences, Brasil) conforme instruções do fabricante e as determinações dos alelos RHCE (RHCE*C, RHCE*c, RHCE*E , RHCE*e), KEL (KEL*01 e KEL*02) e FY (FY*01 , FY*02) foram realizadas por PCR-SSP (Reação em Cadeia da Polimerase – Sequência Específica de Primers). As dificuldades e os motivos de não fenotipagens e das limitações da técnica foram levantadas. As análises estatísticas foram realizadas no SPSS 21.0 (IBM, 2012). **Resultados:** As causas de não fenotipagem inicial foram relacionadas à: anemia falciforme, autoanticorpo e/ou prova de Coombs direto positivo, campo misto e/ou dupla população, prova cruzada incompatível, transfusão recente e causas não descritas. Observou-se que as fenotipagens realizadas previamente, ainda que incompletas, conseguiram definir aproximadamente 50% dos antígenos de grupos analisados: 08 (44,4%) para antígenos RhC/RhE, 09 (50%) para antígeno Kell, Fya e Fyb e 10 (55,6%) para antígenos eritrocitários Rhc e Rhe. Observou-se a concordância com a genotipagem em 57 (96,61%) dos 59 antígenos testados sendo que em 2 (3,38%) casos houve discrepância de resultados entre as técnicas para os alelos RHCE*E e FY*B (FY*02). **Discussão:** A genotipagem foi eficaz na definição