comerciais anti-D (ESD1 BioRad; P3X61, P3X290, PX35, PX21223 B10, RUM-1, ESD-1M–Grifols; D175, D415–Immucor®; NaTH119, LOR-15C9 Imunoscan). As fenotipagens eritrocitárias dos Sistemas Rh (C,c,E,e) e Duffy (Fy*a*, Fy*b*) foram determinadas nas técnicas de Gel (DG Gel Rh Pheno e com antissoros policlonais, Grifols S.A, respectivamente). A genotipagem foi realizada por técnicas de PCR-Multiplex com primers específicos do gene RHD (exons 3, 4, 5, 6, 7 e 9), PCR- RFLP para regiões do gene FY (125G>A e -67T>C), técnicas automatizadas utilizando a plataforma Bloodchip (Kit ID CORE *XT* Grifols S.A.) e sequenciamento Sanger com análise dos 10 exons do gene RHD. **Resultados:** A tipagem RHD apresentou resultado discrepante entre os dois anti-D nos testes de rotina (Anti-D IgM negativo e IgG fracamente positivo). Os demais antissoros apresentaram reações sorológicas fracas e inconsistentes. Além desse achado, a presença de neutropenia leve no hemograma do doador motivou a investigação dos fenótipos dos Sistemas RH e FY: RH:2,-3,4,5 (Ccee) e FY:-1,-2/Fy(a-b-). O PCR-multiplex detectou todos os éxons do gene RHD. As genotipagens, convencional e automatizada confirmaram o polimorfismo GATA em *homozigose* (FY*2_67C>C) na região promotora gene FY, e o sequenciamento apontou a troca de um único nucleotídeo (833G>A) no éxon 6 confirmando o alelo RHD*01W.38. **Discussão:** O fenótipo D fraco tipo 38 está envolvido na aloimunização anti-D, sua prevalência é 1,5% em indivíduos caucasianos de origem portuguesa e o genótipo GATA é relativamente comum em afrodecendentes. A amplificação dos exons RHD, a associação com o haplótipo RHCE*Ce (frequente em caucasianos) e a mutação GATA, foram fatores que determinaram a análise do DNA desse doador pela técnica do Sequeciamento, uma vez que, protocolos de genotipagem estendidos para as variantes RHD consomem tempo e são de alto custo. **Conclusão:** A identificação de indivíduos com fenótipos incomuns é importante tanto para fins transfusionais, como para caracterizar frequências populacionais onde a miscigenação é alta, ou em populações onde normalmente elas não seriam pesquisadas. A disponibilidade de técnicas de sequeciamento proporciona resultados mais acertivos e rápidos uma vez que analisam regiões de exons e introns do gene RHD, onde metodologias de PCRs convencionais demorariam mais ou até mesmo, mostrariam-se inconclusivas.

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EFFECTS OF PROPHYLACTIC RED BLOOD CELL (RBC) TRANSFUSION WITH EXTENDED ANTIGEN MATCHING ON ALLOIMMUNIZATION IN PATIENTS WITH SICKLE CELL DISEASE (SCD)

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Background and aims: As the alloimmunization remains a major complication associated with RBC transfusions in SCD patients and can be associated with Delayed Hemolytic Transfusion Reactions (DHTRs), this study was undertaken to assess the effects of prophylactic RBC transfusion with extended RBC antigen matching on alloimmunization in patients with SCD. **Methods:** A 20-year retrospective study involving 179 patients with SCD transfused with RBCS matched for D, C, c, E, e , K, Fy*a*/Fy*b*, Jk*a*/Jk*b* and S antigens was performed. Data from medical records were evaluated, such as the number of transfusions and the results of immunohematological tests, obtained through serological and molecular techniques. According to the history of the transfusion service, the alloimmunization rate prior to the implementation of the transfusion protocol with extended phenotype matching was 47%. Unexpected antibodies to the Rh system, meaning anti-Rh antibodies in patients whose serologic phenotype was Rh positive, were investigated by molecular genotyping for RH variant alleles. **Results:** During the study period, 42 (23.5%) patients developed red cell antibodies and 137 (76.5%) did not. Among the 42 patients who alloimmunized, 13 (31%) had Rh variants, and in 8 (19%), the variant found would justify the formation of the identified Rh antibody, and 16 (38.1%) patients developed antibodies against antigens of low prevalence. **Discussion:** The provision of antigen-negative blood to patients under chronic transfusions is being recommended to decrease the risk of hemolytic transfusion reactions and to prevent new instances of alloimmunization. Our results showed that patients with SCA benefit from transfusions with extended matching, as demonstrated by the reduction in alloimmunization rates from 47% to 23.5%. We observed in this retrospective analysis that some patients who received external transfusions developed antibodies against the antigens that were being matched, which could be one of the reasons that would justify the rate of alloimmunization found. Another cause may be genotype-discordant phenotype, as some patients may have been phenotyped after recent transfusions before being genotyped. This cohort had a wide variation in exposure to red blood cell transfusions and patients who alloimmunized for Rh and low prevalence antigens were the most exposed, confirming that the number of transfusions influences the alloimmunization rate. Our findings also suggest that RH diversity in patients with SCD and the presence of specific low prevalence antigens in the donor population necessitate an alternative approach to improve RBC matching. **Conclusion:** Our results demonstrate that prophylactic extended matching of RBC antigens significantly reduces alloimmunization rates in transfused patients with SCD, providing safer transfusions and should be considered to all patients on chronic transfusions. This strategy can be even more beneficial if patients with recent transfusions are genotyped, if matching for Rh variants is performed, and if there is greater control of external transfusions.

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ANTI-D ALLOIMMUNIZATION IN SICKLE CELL DISEASE (SCD) PATIENTS WITH WEAK RHD VARIANT ALLELES

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