Original article

When the available blood supply mismatches the needs of the patient

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Background - Substantial regional differences in the genetic patterns related to blood group have been observed across different continents. This diversity means that the blood supply, as an essential part of patient care, is increasingly impacted by global migration. Consequently, the Austrian blood donor population does not match the immigrant patient population. This mismatch is likely to result in the formation of alloantibodies to red cell antigens in the chronically transfused. Subsequently, major difficulties in providing compatible blood emerge.

Material and methods - The study included patients of African origin (n=290) and Caucasians who represent the Austrian donor population (n=1,017). Genetic typing was performed for up to 69 blood group polymorphisms with a multiplex sequence specific primer-PCR including high frequency antigens and antigens for which antisera are not commercially available. By assessing differences in antigen frequencies between the two populations, and using these data for prophylactic matching, we aim to develop tools to increase the quality of patient care.

Results - Results indicate various and significant differences ($p<0.0001$) in antigen frequencies between African patients and the European donor population, especially in the MNS, Duffy, Knops and Rhesus systems.

Discussion - Our data highlight the importance of matching the donor population to the demographics of the patient population. In addition, it underlines the need to recruit donors of African origin and to focus on the upcoming challenges, such as malaria semi-immunity and a significantly higher rate of infectious disease in this population. It is also recommended to apply extended genetic typing to detect rare blood types, and (cryo)storage of rare blood in national and international rare blood banks. Co-operation with regional blood banks should also be encouraged.

Keywords: *transfusion medicine, differences in antigen frequencies, genotyping.*

INTRODUCTION

People have been migrating across the world for centuries. However, the resulting challenges associated with ethnic diversity and the need to ensure an adequate blood supply are a recent development. The human blood group systems show varying

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phenotypes among diverse populations and ethnic groups worldwide. Some may have resulted from the protective effects of certain erythrocyte membrane characteristics against infectious diseases such as malaria[1,2].

The need for donor blood is higher in the African population, as up to 18.2% of the African population carries a significant hemoglobin gene variant, such as HbS, HbC, HbE, HbD, β thalassemia, or α° thalassemia[1-3]. In addition, the formation of alloantibodies after transfusion is higher in patients with hemoglobinopathies than in other patient groups[4-6].

The practice and extent of prophylactic antigen matching to prevent alloimmunization, especially for chronic transfusion-dependent patients, vary widely across institutes and countries. In studies in the UK, the US, and Kuwait the frequency of patients with hemoglobinopathies who produce alloantibodies ranges from 18-76% when only ABO and RhD matching is performed. When additional limited phenotype matching for the antigens C, E, and K is performed, the frequency of alloantibody formation drops to 5-11%, which in turn drops further to 0-7% when extended minor RBC antigen matching is performed[7-16].

In contrast, the frequency of alloimmunization in sickle cell disease (SCD) patients is lower in countries like Jamaica and Uganda, where only ABO and D matches, with rates of 2.6% and 6.1%, respectively. This discrepancy is most likely the result of the homogeneity of recipients and donors in the Jamaican and Ugandan studies[10,17].

To a great extent, the risk for alloimmunization in SCD patients depends on the transfusion regime. Transfusion in an acute setting (e.g., acute chest syndrome) or during an inflammatory state in the recipient, as well as during viral infections, results in an increased rate of alloantibody formation. Recent data also show that alloimmunization rates are higher in single unit packed red blood concentrate transfusion schemes than when red cell exchange therapy with up to 10 pRBCs is utilized[18-23].

In times of increased migration, the risk of alloimmunization becomes a serious concern as the number of patients in need of blood products with phenotypes that do not match those of the donor increases. In this context, due to their immunogenicity and their varying frequency among the ethnic groups, certain blood group antigens are of particular interest and will be discussed in this article.

This study aimed to genetically type individuals with a history of migration for 69 blood group polymorphisms, the objective being to optimize the blood supply and prevent alloantibody formation after multiple transfusions in these patients. The differences in blood group antigen frequencies between African patients (group A) and a donor population (almost exclusively Caucasian; group B), including the resulting challenges, were also investigated. The results of these genetic antigen profiles could promote a more thorough understanding of the differences in frequencies of RBC antigens related to the ethnic origin of patients and provide supplementary information to routine antibody screening.

MATERIALS AND METHODS

The Ethics Committee of the Austrian Red Cross approved this study (20210823_07). Data from routine genotyping of patients were analyzed retrospectively.

DNA isolation

DNA was purified from EDTA-anticoagulated whole blood samples using the Maxwell 16 device and the Maxwell 16 Blood DNA kit (Promega, Fitchburg, USA) according to the manufacturers' instructions.

Genotyping

Fifty-nine out of 63 polymorphisms of the data set "MX3" were included in this analysis. The D exons and d-hybridbox were not included because of a lack of detailed typing. The RHD-pseudogene (D-psi) was included. Genotyping of 63 blood group polymorphisms ("MX3") was performed with an updated version of the multiplex sequence specific primer-PCR (SSP-PCR), as described previously[24]. Primers are listed in *Supplementary Content*, **Table SI**.

Multiplexed SSP-PCRs were carried out in 16 µL reactions using the following conditions:

- 1.35x buffer (Promega), 4.17 mM $MgCl_2$ (Promega), 0.2 mM dNTPs (Thermo Fisher Scientific, Rochester, NY, USA), 0.15-0.95 µM gene specific primers, 0.52 units GoTaq G2 Hot Start Polymerase (Promega), and 2 µL genomic DNA per sample were used.

PCR program (the same for all performed SSP-PCR):

- 94°C 300", 6 cycles (94°C 30", 67°C 40" –0.5°C/cycle, 72°C 110"), 27 cycles (94°C 30", 64°C 40", 72°C 110"),1 hold 72°C 120".

Additional screening was performed for antigens Aua (LU18), Aub (LU19), KCAM (KN9), KDAS (KN10), and Yka

(KN5) named "AKY" (6 polymorphisms). Primers are listed in *Supplementary Content*, **Table SII**.

SSP-PCRs were carried out in 16 µL reactions using the following conditions:

1.0x buffer (Promega), 4.17 mM $MgCl_2$ (Promega), 0.2 mM dNTPs (Thermo Fisher), 0.2-0.64 µM gene specific primers, 0.25 µM 1kb internal control primers, 0.52 units GoTaq G2 Flexi Polymerase (Promega), and 2 µL genomic DNA per sample was used.

PCR products from both reactions were separated by gel electrophoresis using 1.5% agarose gel in TBE, stained with SYBR-Safe DNA Gel Stain (Invitrogen, Waltham, MA, USA).

Statistical analysis

Statistical calculations were performed using the "N-1" χ^2 test, as recommended by Campbell (2007) and Richardson (2011), using MedCalc (Version 20.027) software (MedCalc Software Ltd, Ostend, Belgium).

For original data, please contact the Corresponding Author.

RESULTS

A total number of 1,307 patients of African (group A=290) and Caucasian (group B=1,017) origin were genotyped for expression patterns with 63 different primer pairs. Out of these, 224 from group A and 272 from group B were typed with the primer set "AKY" for 5 additional antigens. **Table I** and **Table II** present allele frequencies of group A samples compared to allele frequencies of group B samples.

No significant differences were observed for LU1, LU2, LU8 and LU14, K11 and K17, DI1, DI2, DI3 and DI4, YT1 and YT2, SC1 and SC2, CO1 and CO2, LW5 and LW7, CH/RG, IN1 and IN2, JR1 and VEL1.

Rh blood group system

Group A samples showed a 5.52% presence of RHD-pseudogene (D-psi), compared to 0.093% (1 of 1,017) of Caucasian donor samples (Group B).

Marked differences in frequency were observed with regard to the C, c, E, and C^W antigens among the two ethnic groups. Positivity rates of 17.93% in group A and 67.42% in group B for C (RH2) and 96.21% (group A) and 80.04% (group B) for c (RH4) were found. The single nucleotide polymorphism (SNP) for E (RH3) was detected in 9.66% of group A samples and 23.40% of group B, whereas comparable rates for the antigen e (RH5) were obtained in both groups. The SNP for the antigen C^w (RH8) antigen was not found in group A; however, it was detected in 3.74% of group B subjects.

Duffy

Frequencies of the Duffy alleles for group B are 65.88% and 78.56% for FY1 and FY2, respectively, with predicted phenotype frequencies of 0.20% Fy(a–b–), 44.64% Fy(a+b+), 33.92% Fy(a–b+), and 21.24% Fy(a+b–). In contrast, in group A samples, the FY^{ES} allele reached a frequency as high as 89.66% with rates of 9.66% for FY1 and 15.86% for FY2, which results in predicted phenotypes of 78.28% Fy(a–b–), 3.79% Fy(a+b+), 12.07% Fy(a–b+), and 5.86% Fy(a+b–).

Knops and high-frequency antigens

Further differences were observed in the Knops blood group system, such as the KN4 (Sl^a) allele with positive rates of 99.90% in group B and in 67.24% of group A. In addition, the SNP for KN3 (McC^a) was detected in 100% of group B samples and was absent in 3.10% of group A. The SNP for KN1 (Kn^a) was detected at high frequencies in both groups.

Moreover, different frequencies for the SNP for KCAM (41.52% group A, 93.38% group B) and the antithetical antigen KDAS (86.61% group A, 52.57% group B) were observed. KN5 (Yk^a) differs slightly between the groups and was less common in group B (93.75 *vs* 98.21%). Within the Kell system, the KEL1 (K) allele was more prevalent in group B (10.13%) than in group A (1.72%). The KEL2 (k) negative genotype was not detected in group A but found in 1.18% of group B individuals. KEL6 (Js^a) was practically absent in group B (0.20%) but found in 12.41% of group A samples. The antithetical KEL7 (Js^b) is considered a high frequency antigen in both populations, with 100% positive in group B and 99.31% in group A.

MNSs

The two groups showed similar frequencies of most of the tested alleles of the MNS blood group system. The M (MNS1) allele was found in 72.41% of group A and 77.97% of group B samples. The N (MNS2) allele had similar frequencies with 76.90% group A and 71.29% group B samples. The s (MNS4) allele was detected in 89.66% (group A) and 88.99% (group B). Some difference was seen regarding the allele frequency for S (MNS3; 32.07% in group A and 49.16% in group B) and the allele referring

Table I - *Allele frequencies of antigens with relevant differences between group A and group B*

Antigens	Positive - group A N=290	Positive - group B N=1,017	% difference
RH			
D-psi	5.52%	0.10%	5.42% (p<0.0001)
C (RH2)	17.93%	67.42%	49.49% (p<0.0001)
c (RH4)	96.21%	80.04%	16.17% (p<0.0001)
E (RH3)	9.66%	23.40%	13.74% (p<0.0001)
e (RH5)	98.97%	97.25%	1.72% (p=0.0894)
C^w (RH8)	0.00%	3.74%	3.74% (p=0.0008)
FY			
FY1 (Fy^a)	9.66%	65.88%	56.22% (p<0.0001)
FY2 (Fy^b)	15.86%	78.56%	62.70% (p<0.0001)
FY ^{ES}	89.66%	1.57%	88.09% (p<0.0001)
Predicted Fy phenotype frequencies			
Fya+b+	3.79%	44.64%	40.85% (p<0.0001)
Fya+b-	5.86%	21.24%	15.38% (p<0.0001)
Fya-b+	12.07%	33.92%	21.85% (p<0.0001)
Fynull	78.28%	0.20%	78.08% (p<0.0001)
KN			
KN1 (Kn^a)	99.66%	99.31%	0.35% (p=0.5005)
KN3 (McC^a)	96.90%	100.00%	3.10% (p<0.0001)
KN4 (Sl^a)	67.24%	99.90%	32.66% (p<0.0001)
KEL			
KEL1 (K)	1.72%	10.13%	8.41% (p<0.0001)
KEL2 (k)	100.00%	98.82%	1.18% (p =0.0632)
KEL (Kp^a)	0.34%	2.78%	2.44% (p=0.0133)
KEL (Kp^b)	100.00%	99.89%	0.11% (p=0.5722)
KEL6 (Js^a)	12.41%	0.20%	12.21% (p<0.0001)
KEL7 (Js^b)	99.31	100.00%	0.69% (p=0.0080)
MNS - GYPA, GYPB			
MNS1 (M)	72.41%	77.97%	5.56% (p=0.0482)
MNS2 (N)	76.90%	71.29%	5.61% (p=0.0591)
MNS3 (S)	32.07%	49.16%	17.09% (p<0.0001)
MNS4 (s)	89.66%	88.99%	0.67% (p=0.7464)
MNS5 (U)	97.59%	100.00%	2.41% (p<0.0001)
$U+^w$	1.03%	0.00%	1.03% (p=0.0012)
U negative	1.38%	0.00%	1.38%(p=0.0002)
DO			
DO1 (Doa)	42.07%	62.14%	20.07% (p< 0.0001)
DO2 (Dob)	90.00%	84.76%	5.24% (p=0.0237)
DO4 (Hy)	98.97%	100.00%	1.03% (p=0.0012)
DO5 (Joa)	100.00%	100.00%	
JK			
JK1(Jka)	97.76%	71.78%	25.98% (p<0.0001)
JK2 (Jkb)	50.00%	74.14%	24.14% (p<0.0001)

Table II - *Additionally typed antigens in Knops and Lutheran system*

Antigens	Positive group A n=224	Positive group B n=272	% difference
KN			
KN5 (Yka)	98.21%	93.75%	4.46% (p=0.0142)
KN9 (KCAM)	41.52%	93.38%	51.86% (p<0.0001)
KN10 (KDAS)	86.61%	52.57%	34.04% (p<0.0001)
LU			
LU18 (Au^a)	80.80%	93.75%	12.95% (p<0.0001)
LU19 (Au^b)	65.63%	53.68%	11.95% (p=0.0071)

to the expression of the antigen U (MNS5). The allele was found in 100% of individuals in group B but was absent in 1.38%, and variant in 1.03% of group A samples.

Dombrock

In the Dombrock blood group system, a predominance of the Do^b (DO2) allele in the samples in both groups was observed (90.00% in group A and 84.76% in group B). The Do^a (DO1) allele was detected in 42.07% of group A samples and of 62.14% of group B samples.

Kidd

Interestingly, differences were also identified regarding the Kidd blood group system, with positivity rates of 97.76% (group A) and 71.78% (group B) for Jk^a (JK1) and 50.00% (group A) and 74.14% (group B) for Jk^b (JK2).

DISCUSSION

Our results indicate significant differences in antigen frequencies between African patients and the European donor population, in particular with regard to the MNS, Duffy, Knops, and Rh systems. Some rare blood types were found exclusively in the African patient group, for example, Fy_{null}, Js^b negative, and U negative. The Fy^a and Fy^b antigens are especially relevant, as these are almost always expressed amongst individuals of most ethnicities while the Fy_{null} phenotype is exceedingly rare. The Fy_{null} phenotype was present in 78.28% of African patients typed in this study, which could potentially result in the formation of anti-Fy3. As a result of the mismatch between donor and patient populations, some blood centers have developed guidelines by which Fy_{null} patients are preferably transfused with Fy^a negative blood to prevent or minimize alloimmunization[25]. Therefore, the importance of broad donor typing and prophylactic matching is highlighted, especially given that 65.88% of our group B samples (donor population) expresses the Fy^a antigen. Nevertheless, most transfusion services will experience major problems in providing suitable packed red blood cells for patients with anti-Fy3 or other antibody specificities against HFAs, as compatible blood can only be found in donors of African origin.

Patients and potential donors of African origin should receive extended typing in order to identify rare blood types; this should include high prevalence antigens. Ideally, this should be performed genetically, as serologic methods are limited by high cost and the restricted availability of antisera. These data can also be used for prophylactic antigen matching, to help identify alloantibodies, and assist in the search for antigen-negative blood for antibody carriers.

It is vital that national blood centers develop and implement strategies to encourage donations from ethnic minorities. Once again, this emphasizes the need to actively recruit and maintain donors of African origin[24,26-30].

However, transfusion services will have to overcome several challenges before admitting potential donors of African origin to the donor population. These include the stipulation of national exclusion criteria or policies related to malaria, as well as the modification of testing algorithms, as these populations tend to have significantly higher rates of infectious diseases. We agree with Berzuini *et al.* that the proposed extensive testing for malaria, Hepatitis B core antibody (Anti-HBc), and for the exclusion of hemoglobinopathies in this donor group would be appropriate[31].

Malaria testing can be extremely challenging as there is still no gold standard available. In an Italian study, up to 72% of potential donors of African origin tested positive for malarial antibodies. Asymptomatic, semi-immune donors pose a substantial risk for transfusion-transmitted malaria; indeed, Assennato *et al.* were able to detect plasmodial DNA in 3.6% of the antibody carriers[33]. However, chronic infection with parasitemia, especially with *P. ovale* and *P. malariae*, in the at risk population can be missed by malaria antibody tests that use *P. falciparum* and *P. vivax* antigens as targets[32,33].

Some countries like the UK, Denmark, Finland, and New Zealand have selective malaria antibody screening in combination with nucleic acid amplification testing (NAT)

in place to allow such donors to donate. Other countries such as Norway and Spain use selective testing for malaria antibodies to accept new donors or to reduce the deferral time. In Brazil, a malarial testing algorithm was implemented for middle- and low-risk donors of endemic regions[34,35].

Rare blood banks need to develop protocols and policies on how rare donors and the respective rare blood units, with either positive malaria antibodies or isolated anti-HBc positivity, are managed[31].

To optimize the blood supply for anti-HFA antibody carriers, the (cryo)storage and distribution of rare blood units through national and international rare blood banks is once again highly recommended[31]. In Europe, African donor blood is often rare; therefore, centralization of rare donor databanks, storage of red blood units, and possibly even antibody identification in these centers can assist national and internal collaboration, particularly when blood units are required urgently.

Antigen variability between ethnic groups can also be challenging with regards to antibody screening and identification. A handful of countries have customized their routine testing algorithm, due to differences in antigen frequencies and consequently in alloantibodies, in order to fulfil the needs of their population[36,37]. As such, some manufacturers offer specific red cell antibody screening panels that cater for certain populations. Therefore, extended typing of red blood cells from patients with African origin and collection thereof for antibody identification is especially important for reference laboratories.

CONCLUSIONS

The data obtained from this study, proves once again that there is a great need for donors who match the patient population. This is especially true for chronically transfused patients. We are still unable to provide a 100% match between patients and donors, and the formation of alloantibodies will remain a transfusion-associated risk until this can be achieved.

Transfusion services should take additional measures in order to establish state-of-the-art blood supply in countries that face these challenges of global migration, or at least form collaborations with national or international services and reference laboratories that can provide support.

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AUTHORSHIP CONTRIBUTIONS

LW and CJ designed the study. WA, ES and CJ analyzed and validated the samples. VW provided samples and patient information. CP, LW, VW and WA evaluated and interpreted the data. LW, CP, ER and WA wrote the manuscript. All Authors critically revised and approved the final version for publication.

The Authors declare no conflicts of interest.

REFERENCES

1. Owaidah AY, Naffaa NM, Alumran A, Alzahrani F. Phenotype frequencies of major blood group systems (Rh, Kell, Kidd, Duffy, MNS, P, Lewis, and Lutheran) among blood donors in the eastern region of Saudi Arabia. J Blood Med 2020; 11: 59-65. doi: 10.2147/JBM.S236834.
2. Cyrklaff M, Sanchez CP, Kilian N, Bisseye C, Simpore J, Frischknecht F, et al. Hemoglobins S and C interfere with actin remodeling in Plasmodium falciparum-infected erythrocytes. Science 2011; 334: 1283-1286. doi: 10.1126/science.1213775.
3. Modell B, Darlison M. Global epidemiology of haemoglobin disorders and derived service indicators. Bull World Health Organ 2008; 86: 480-487. doi: 10.2471/blt.06.036673.
4. Perseghin P, Balduzzi A, Galimberti S, Dassi M, Baldini V, Valsecchi MG, et al. Red blood cell support and alloimmunization rate against erythrocyte antigens in patients undergoing hematopoietic stem cell transplantation. Bone Marrow Transplant 2003; 32: 231-236. doi: 10.1038/sj.bmt.1704114.
5. Campbell-Lee SA. Red blood cell alloimmunization in sickle cell anemia: more questions than answers? Transfusion 2012; 52: 214-216. doi: 10.1111/j.1537-2995.2011.03502.x.
6. Ameen R, Al-Shemmari S, Al-Humood S, Chowdhury RI, Al-Eyaadi O, Al-Bashir A. RBC alloimmunization and autoimmunization among transfusion-dependent Arab thalassemia patients. Transfusion 2003; 43: 1604-1610. doi: 10.1046/j.1537-2995.2003.00549.x.
7. Ambruso DR, Alcorn R, Dixon DJ, Brown LJ, Vaughn WM, Hays T. Experience with donors matched for minor blood group antigens in patients with sickle cell anemia who are receiving chronic transfusion therapy. Transfusion 1987; 27: 94-98. doi: 10.1046/j.1537-2995.1987.27187121485.x.
8. Rosse W, Gallagher D, Kinney T, Castro O, Dosik H, Moohr J, et al. Transfusion and alloimmunization in sickle cell disease. The Cooperative Study of Sickle Cell Disease. Blood 1990; 76: 1431-1437. doi: 10.1182/blood.v76.7.1431.bloodjournal7671431.
9. Vichinsky EP, Luban NLC, Wright E, Olivieri N, Driscoll C, Pegelow CH, et al. Prospective RBC phenotype matching ina stroke-Prevention trial in sickle cell anemia: A multicenter transfusion trial. Transfusion 2001; 41: 1086-1092. doi: 10.1046/j.1537-2995.2001.41091086.x.

10. Olujohungbe A, Hambleton I, Stephens L, Serjeant B, Serjeant G. Red cell antibodies in patients with homozygous sickle cell disease: a comparison of patients in Jamaica and the United Kingdom. Br J Haematol 2001; 113: 661-665. doi: 10.1046/j.1365-2141.2001.02819.x.
11. Aygun B, Padmanabhan S, Paley C, Chandrasekaran V. Clinical significance of RBC alloantibodies and autoantibodies in sickle cell patients who received transfusions. Transfusion 2002; 42: 37-43. doi: 10.1046/j.1537-2995.2002.00007.x.
12. Castro O, Sandler SG, Houston-Yu P, Rana S. Predicting the effect of transfusing only phenotype-matched RBCs to patients with sickle cell disease: theoretical and practical implications. Transfusion 2002; 42: 684-690. doi: 10.1046/j.1537-2995.2002.00126.x.
13. Ameen R, Al-Shemmari S, Al-Humood S, Chowdhury RI, Al-Eyaadi O, Al-Bashir A. RBC alloimmunization and autoimmunization among transfusion-dependent Arab thalassemia patients. Transfusion 2003; 43: 1604-1610. doi: 10.1046/j.1537-2995.2003.00549.x.
14. Sakhalkar VS, Roberts K, Hawthorne LM, McCaskill DM, Veillon DM, Caldito GG, et al. Allosensitization in patients receiving multiple blood transfusions. Ann N Y Acad Sci 2005; 1054: 495-499. doi: 10.1196/annals.1345.072.
15. Davies SC, McWilliam AC, Hewitt PE, Devenish A, Brozovic M. Red cell alloimmunization in sickle cell disease. Br J Haematol 1986; 63: 241-245. doi: 10.1111/j.1365-2141.1986.tb05546.x.
16. Michail-Merianou V, Pamphili-Panousopoulou L, Piperi-Lowes L, Pelegrinis E, Karaklis A. Alloimmunization to red cell antigens in thalassemia: comparative study of usual versus better-match transfusion programmes. Vox Sang 1987; 52: 95-98. doi: 10.1111/j.1423-0410.1987.tb02999.x.
17. Natukunda B, Schonewille H, Ndugwa C, Brand A. Red blood cell alloimmunization in sickle cell disease patients in Uganda. Transfusion 2010; 50: 20-25. doi: 10.1111/j.1537-2995.2009.02435.x.
18. Tormey CA, Hendrickson JE. Transfusion-related red blood cell alloantibodies: Induction and consequences. Blood 2019; 133: 1821-1830. doi: 10.1182/blood-2018-08-833962.
19. Evers D, Middelburg RA, Haas M de, Zalpuri S, Vooght KMK de, Kerkhof D van de, et al. Red-blood-cell alloimmunisation in relation to antigens' exposure and their immunogenicity: a cohort study. Lancet Haematol 2016; 3: e284-292. doi: 10.1016/S2352-3026(16)30019-9.
20. Evers D, van der Bom JG, Tijmensen J, Middelburg RA, de Haas M, Zalpuri S, et al. Red cell alloimmunisation in patients with different types of infections. Br J Haematol 2016; 175: 956-966. doi: 10.1111/bjh.14307.
21. Celli R, Schulz W, Hendrickson JE, Tormey CA. A novel network analysis tool to identify relationships between disease states and risks for red blood cell alloimmunization. Vox Sang 2017; 112: 469-472. doi: 10.1111/vox.12515.
22. Ryder AB, Hendrickson JE, Tormey CA. Chronic inflammatory autoimmune disorders are a risk factor for red blood cell alloimmunization. Br J Haematol 2016; 174: 483-485. doi: 10.1111/bjh.13781.
23. Fasano RM, Booth GS, Miles MR, Du L, Koyama T, Meier ER, et al. Red Blood Cell Alloimmunization Is Influenced By Recipient Inflammatory State At Time Of Transfusion In Patients With Sickle Cell Disease. Br J Haematol 2015; 168: 291-300. doi: 10.1111/bjh.13123.
24. Jungbauer C. Routine use of DNA testing for red cell antigens in blood centres. Transfus Apher Sci 2011; 45: 61-68. doi: 10.1016/j.transci.2011.06.004.
25. Tournamille C, Colin Y, Cartron JP, Le Van Kim C. Disruption of a GATA motif in the Duffy gene promoter abolishes erythroid gene expression in Duffy-negative individuals. Nat Genet 1995; 10: 224-228. doi: 10.1038/ng0695-224.
26. Flesch BK, Scherer V, Just B, Opitz A, Ochmann O, Janson A, et al. Molecular blood group screening in donors from Arabian Countries and Iran using high-throughput MALDI-TOF mass spectrometry and PCR-SSP. Transfus Med Hemother 2020; 47: 396-408. doi: 10.1159/000505495.
27. Flegel WA, Gottschall JL, Denomme GA. Integration of red cell genotyping into the blood supply chain: a population-based study. Lancet Haematol 2015; 2: e282-289. doi: 10.1016/S2352-3026(15)00090-3.
28. Karafin MS, Field JJ, Gottschall JL, Denomme GA. Barriers to using molecularly typed minority red blood cell donors in support of chronically transfused adult patients with sickle cell disease. Transfusion 2015; 55: 1399-1406. doi: 10.1111/trf.13037.
29. Thompson AA. Ideal donors, imperfect results in sickle cell disease. Blood 2013; 122: 858-859. doi: 10.1182/blood-2013-06-507368.
30. Klinkenberg EF, Fransen MP, de Kort WLAM, van Weert JCM, Huis in't Veld EMJ. Blood donation among individuals of African origin in the Netherlands: how are barriers and motivators associated with intention? Blood Transfus 2021; 19: 24-33. doi: 10.2450/2020.0049-20.
31. Berzuini A, Foglieni B, Spreafico M, Gerosa A, Revelli N, Paccapelo C, et al. Black blood matters. Blood Transfus 2021; 19: 363-365. doi: 10.2450/2021.0144-21.
32. Seed CR, Cheng A, Davis TME, Bolton WV, Keller AJ, Kitchen A, et al. The efficacy of a malarial antibody enzyme immunoassay for establishing the reinstatement status of blood donors potentially exposed to malaria. Vox Sang 2005; 88: 98-106. doi: 10.1111/j.1423-0410.2005.00605.x.
33. Assennato SM, Berzuini A, Foglieni B, Spreafico M, Allain J-P, Prati D. Plasmodium genome in blood donors at risk for malaria after several years of residence in Italy. Transfusion 2014; 54: 2419-2424. doi: 10.1111/trf.12650.
34. Reesink HW, Panzer S, Wendel S, Levi JE, Ullum H, Ekblom-Kullberg S, et al. The use of malaria antibody tests in the prevention of transfusion-transmitted malaria. Vox Sang 2010; 98: 468-478. doi: 10.1111/j.1423-0410.2009.01301.x.
35. Hoogen LL van den, Bareng P, Alves J, Reyes R, Macalinao M, Rodrigues JM, et al. Comparison of commercial ELISA kits to confirm the absence of transmission in malaria elimination settings. Front Public Health 2020; 8: 480. doi: 10.3389/fpubh.2020.00480.
36. Park TS, Oh SH, Choi JC, Lee DD, Kim HH, Chang CL, et al. The clinical significance of antibody screening test including Dia+ panel cell in Asian-Mongoloid populations. J Korean Med Sci 2003; 18: 669-672. doi: 10.3346/jkms.2003.18.5.669.
37. Lin M. Elimination of pretransfusion RhD typing at Mackay Memorial Hospital, Taiwan-30-year experience (1988-2017). Vox Sang 2021; 116: 234-238. doi: 10.1111/vox.13000.