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In-Depth Study of ABO, Rhesus, Kell, Duffy, MNS, and Kidd Red Cell Phenotypes in a Cohort of Sickle Cell Patients in Lubumbashi, Democratic Republic of the Congo

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ABSTRACT

Introduction

In the Democratic Republic of Congo (DRC), transfusion management for patients with sickle cell disease (SCD) is traditionally limited to ABO and RH1 (D) compatibility screening. This practice exposes multi-transfused pediatric populations to a high hidden risk of red blood cell (RBC) alloimmunization due to unmonitored minor blood group polymorphisms.

Purpose

To establish extended immunohematological mapping of the Rh, Kell, Duffy, Kidd, and MNS systems in a pediatric SCD cohort in Lubumbashi, DRC.

Methods

A cross-sectional study was conducted involving 98 pediatric SCD patients managed across specialized urban medical facilities in Lubumbashi. Extended erythrocyte antigen phenotyping was performed using standard hemagglutination techniques. Statistical analyses were executed using IBM SPSS Statistics (Version 20.0).

Results

The cohort had a mean age of 4.26 ± 3.07 years and a high clinical transfusion burden, with 40.8% of subjects having received three or more transfusions. Complete antigenic invariance (100.0% prevalence) was observed for D, c, e, and s antigens. Conversely, phenotypic variations were identified in antigens C (11.2%) and E (7.1%). The ABO system is dominated by group O (57.14%). The protective Duffy-null phenotype Fy(a−b−) predominated (89.8%), whereas the Kidd system revealed a high proportion of the Jk(b−) variant (66.3%). Bivariate analyses confirmed that the distribution of these blood group variations was statistically independent of sex and geographic origin ($p > .05$).

Conclusion

Current national transfusion guidelines expose multi-transfused children to significant, unaddressed immunological risk. Implementing extended phenotyping and regional donor-matching registries is necessary to mitigate clinical alloimmunization complications in the DRC.

INTRODUCTION

Sickle cell disease (SCD) remains the most prevalent genetic disorder and a public health burden in sub-Saharan Africa (Chou et al., 2020; Diallo, 2008). In this region, clinical management relies on blood transfusion therapy to manage decompensated chronic anemia and treat severe vaso-occlusive crises, acute chest syndromes, and splenic sequestrations (Novelli & Gladwin, 2016). However, repeated transfusion exposes polytransfused patients to anti-erythrocyte alloimmunization (Aygun et al., 2002; Pirenne et al., 2023). This immunological complication stems from human blood group system polymorphism, characterized by antigenic variability between donors and recipients (O'Suoji et al., 2013).

While the ABO system and the RH1 (D) antigen form the basis for standard transfusion safety (Flegel, 2006; Italian, 2021), other immunogenic systems play a critical role in irregular alloantibody-mediated hemolytic events (Harmening, 2018). The extended Rhesus system (antigens C, c, E, e) and the Kell system (KEL) receive attention due to their immunogenicity (Barro et al., 2018; Chiaroni et al., 2024). Minor systems such as Duffy (Fy^a, Fy^b), Kidd (Jk^a, Jk^b), and MNS (M, N, S, s) also induce alloimmunization in patients receiving unphenotyped transfusions (Cohn et al., 2023; Reid et al., 2012).

International bodies, including the American Society of Hematology (ASH), advocate extensive erythrocyte phenotyping before initial transfusion in SCD patients to ensure compatibility beyond ABO-RhD profiles (Chou et al., 2020; Tariq et al., 2022). Despite these guidelines, routine transfusion practice in developing regions remains limited to standard ABO-RhD matching due to resource constraints and limited local epidemiological data (Sawadogo et al., 2023).

Although international literature documents antigenic frequencies among populations of African origin (Howes et al., 2011), data on extended erythrocytic polymorphism in the Democratic Republic of Congo (DRC)—specifically Haut-Katanga Province—remain fragmented. In Lubumbashi, an endemic area for SCD (Katamea, 2024), local phenotypic variant prevalence and alloimmunization risk under unphenotyped transfusion protocols are unquantified. Characterizing local antigenic profiles is necessary to identify high-risk phenotypes, quantify regional demand for phenotyped units, and support targeted blood donor registries (Gadji et al., 2023; Seck et al., 2019).

Objectives

This study aimed to characterize extended phenotypic erythrocyte profiles (ABO, Rhesus, Kell, Duffy, MNS, Kidd) within a pediatric SCD cohort in Lubumbashi. The primary objective was to map regional requirements for minor immunogenic blood group phenotypes.

We hypothesized that the SCD population of Lubumbashi exhibits a high prevalence of antigen-negative phenotypes (Duffy-, Kidd-, and MNS-null profiles), reflecting selective pressures in Central African populations related to malaria endemicity (Howes et al., 2011). We further hypothesized that this local phenotypic pattern contrasts with donor diversity, presenting an unmonitored risk factor for alloimmunization from initial transfusion exposures (Pirenne et al., 2023).

METHODS

Research Framework

This descriptive, cross-sectional study was conducted at the Medical Laboratory of the Lubumbashi University Clinics in Lubumbashi, DRC. As the primary tertiary healthcare center in Haut-Katanga Province, this institution serves a population with high SCD endemicity. The laboratory hematology unit handled specialized pediatric tracking and immunohematological diagnostic workflows.

Study Participants and Recruitment

The target population comprised pediatric SCD patients receiving care across health facilities in Lubumbashi. A sample of 98 patients aged 1–12 years was enrolled (72 males, 26 females).

Participants were selected using non-probability convenience sampling among ambulatory and hospitalized SCD patients across partner health facilities in Lubumbashi. Recruitment was conducted continuously within pediatric and hematology departments until target enrollment was achieved. Legal guardians were contacted during routine consultations or post-transfusion follow-

ups, and inclusion was validated upon obtaining signed informed consent.

Inclusion Criteria

i. Documented diagnosis of SCD confirmed by hemoglobin electrophoresis.

ii. Patient managed within a participating healthcare facility in Lubumbashi.

iii. Provision of written informed consent by legal guardians.

Exclusion Criteria

i. Undocumented SCD diagnosis.

ii. Transfusion within 3 months prior to blood sampling (to prevent donor RBCs from interfering with recipient phenotyping).

iii. Refusal of consent.

Bias Control

1. **Selection Bias:** Recruitment was conducted without restriction based on ethnicity, tribe, or social class, covering seven urban municipalities to promote sample diversity.
2. **Classification Bias:** Interference from donor erythrocytes was controlled by enforcing a 3-month post-transfusion exclusion window.
3. **Measurement Bias:** Macroscopic hemagglutination was evaluated independently by two laboratory technicians in a double-blind manner. Discrepant or ambiguous results were retested immediately.

Data Collection and Laboratory Procedures

Venous blood samples were collected in EDTA tubes and analyzed within 24 hours (stored at 2°C–8°C prior to testing). Immunohematological testing was performed using commercial reagents according to manufacturer protocols:

a) **ABO, Rhesus, and Kell Systems:** Direct macroscopic agglutination was performed on opaline plates at room temperature (18°C–25°C) using DIAGAST reagents (Loos, France; 2026 manufacturing batches).

b) **Duffy, MNS, and Kidd Systems:** Testing was performed using SeraClone reagents (Biotest, Dreieich, Germany). Slide hemagglutination and tube Indirect Antiglobulin Tests (IAT) were used to detect weak antigen expression.

Phenotyping Protocols

A. **Direct Plate Hemagglutination (ABO, Rhesus, Kell):** One drop of monoclonal antiserum (DIAGAST Anti-A, -B, -D, -C, -E, -c, -e, -KEL1, or -KEL2) was combined with one drop of whole blood or a 5%–10% RBC suspension on an opaline plate. The mixture was rotated for 2 minutes prior to immediate visual inspection.

B. **Indirect Antiglobulin Test in Tubes (Duffy, MNS, Kidd):** Two drops of specific SeraClone antiserum were mixed with one drop of a 3%–5% RBC suspension in glass tubes and incubated at 37°C for 15–30 minutes. The cells were washed three times with physiological saline, followed by the addition of two drops of Anti-Human Globulin (AHG). Tubes were centrifuged at 1,000 g for 1 minute and gently resuspended for macroscopic reading.

Interpretation

I. **Positive Reaction:** Macroscopic agglutinates against a clear background.

II. **Negative Reaction:** Homogeneous, smooth erythrocyte suspension without visible aggregates.

Internal Quality Control (IQC)

Analytical quality was maintained using certified control panels (HEMA CQI, DIAGAST/Biotest) to verify lot avidity and reactivity. Auto-controls (patient RBCs tested against autologous serum) were performed to exclude false positives from in vivo auto-agglutination. Reagent buffer controls were used to exclude non-specific agglutination.

Statistical Analysis

Data were logged in Microsoft Excel 2016 and analyzed using IBM SPSS Statistics (Version 20.0). Quantitative variables were reported as mean ± standard deviation (M ± SD), median, and interquartile range (IQR). Qualitative

variables were summarized as absolute frequencies (n) and percentages (%).

Ninety-five percent confidence intervals (95% CIs) for proportions were calculated using the Wilson score method. Bivariate associations were evaluated using Spearman's rank correlation coefficient (ρ), the Mann-Whitney U test, Pearson's chi-square test (χ^2), or Fisher's exact test as appropriate. Statistical significance was defined as $p < .05$.

RESULTS

Inclusion Flow and Cohort Characteristics

A total of 98 patients with confirmed SCD met all criteria and completed extended RBC phenotyping.

[EVALUATION] ---> 98 SCD patients evaluated in Lubumbashi
↓
[INCLUSION] ---> 98 Enrolled (Confirmed SS, post-transfusion window >= 3 months, signed consent)
↓
[ANALYSIS] ---> 98 Samples phenotyped (DIAGAST / Biotest ranges, double-blind reading)
↓
[COHORT] ---> 98 Validated complete phenotypic profiles

Sociodemographic and Clinical Profile

The cohort mean age was 4.26 ± 3.07 years (range: 1–12 years; median: 3.00, IQR: 4.00). Males comprised 73.5% (n = 72) and females 26.5% (n = 26) of the sample.

The cumulative transfusion burden averaged 2.06 ± 0.87 units (median: 2.00, IQR: 2.00). Overall, 34.7% (n = 34) had received 1 transfusion, 24.5% (n = 24) 2 transfusions, and 40.8% (n = 40) ≥ 3 transfusions. Geographical distribution across Lubumbashi's seven municipalities showed higher representation from Annexe (25.5%, n = 25) and Lubumbashi (18.4%, n = 18).

Table 1:
Sociodemographic, Clinical, and Geographic Characteristics of the Cohort (N = 98)

Parameter / Variable	Value / Frequency (n)	Percentage (%) / Summary	95% CI
Anthropometric Parameters			
Age (years), M ± SD	4.26 ± 3.07	–	[3.64, 4.87]
Median age (years)	–	3.00	–
Interquartile Range (IQR)	–	4.00	–
25th Percentile (Q_1)	–	2.00	–
75th Percentile (Q_3)	–	6.00	–
Range (Min / Max)	1 / 12	–	–
Sex			
Male	72	73.5%	[64.1%, 81.1%]
Female	26	26.5%	[18.9%, 35.9%]
Clinical Parameters			
Cumulative Transfusions, M ± SD	2.06 ± 0.87	–	[1.89, 2.24]
Transfusion Categories			
1 Transfusion	34	34.7%	[25.5%, 44.8%]
2 Transfusions	24	24.5%	[16.5%, 34.2%]
≥ 3 Transfusions	40	40.8%	[31.6%, 50.7%]
Geographic Distribution			
Annexe	25	25.5%	[17.7%, 35.2%]
Lubumbashi	18	18.4%	[11.7%, 27.5%]
Katuba	13	13.3%	[7.7%, 21.7%]
Kenya	12	12.2%	[6.9%, 20.4%]
Rwashi	11	11.2%	[6.1%, 19.3%]
Kamalondo	10	10.2%	[5.3%, 18.1%]
Kampemba	9	9.2%	[4.6%, 16.9%]

The ABO Blood Group System

Table 2:
The ABO Blood Group System of the participants

Systems	Phenotypes / Antigens	Frequency (n=98)	Percentage (%)
ABO	Group O	56	57.14
	Group B	30	30.61
	Group A	11	11.22
	Group AB	1	1.02

Based on the ABO system, group O is the most prominent and dominated by 57.14% against group AB, highlighting its usual rarity at 1.02%).

Antigenic and Phenotypic Profiles

Antigenic mapping showed 100.0% positivity for D, c, e, and s. In the Rhesus system, antigen C was detected in 11.2% (n = 11) and antigen E in 7.1% (n = 7). In the Kell system, Kel-1 (K) prevalence was low (2.0%, n = 2), while Kel-2 (k) reached 98.0% (n = 96).

Duffy system antigens showed low prevalence (Fy^a: 5.1%, n = 5; Fy^b: 6.1%, n = 6). The Kidd system demonstrated high positivity for Jk^a (95.9%, n = 94) relative to Jk^b (33.7%, n = 33). In the MNS system, antigen M was present in 24.5% (n = 24) and antigen N in 16.3% (n = 16).

Table 3:
Antigenic Distribution Across Evaluated Blood Group Systems (N = 98)

System	Antigen	Positive (n, %)	Negative (n, %)
Rhesus	D	98 (100.0%)	0 (0.0%)
	C	11 (11.2%)	87 (88.8%)
	E	7 (7.1%)	91 (92.9%)
	c	98 (100.0%)	0 (0.0%)
	e	98 (100.0%)	0 (0.0%)
Kell	Kel-1 (K)	2 (2.0%)	96 (98.0%)
	Kel-2 (k)	96 (98.0%)	2 (2.0%)
Duffy	Fy^a	5 (5.1%)	93 (94.9%)
	Fy^b	6 (6.1%)	92 (93.9%)
Kidd	Jk^a	94 (95.9%)	4 (4.1%)
	Jk^b	33 (33.7%)	65 (66.3%)

MNS	M	24 (24.5%)	74 (75.5%)
	N	16 (16.3%)	82 (83.7%)
	S	9 (9.2%)	89 (90.8%)
	s	98 (100.0%)	0 (0.0%)

Phenotype analysis confirmed that D + C - E - c + e + was the predominant Rhesus profile (81.6%, n = 80). The Duffy-null phenotype Fy(a- b-) accounted for 89.8% (n = 88). The Kidd system was led by Jk(a+b-) at 64.3% (n = 63), and Kell was dominated by K - k+ at 98.0% (n = 96).

Table 4:
Distribution of Complex Erythrocyte Phenotypes (N = 98)

System	Phenotypic Profile	Count (n)	Percentage (%)	95% CI (Wilson)
Rhesus	D+C-E-c+e+	80	81.6%	[72.9%, 88.0%]
	D+C-E-c+e+	11	11.2%	[6.4%, 18.9%]
	D+C-E+c+e+	7	7.1%	[3.5%, 13.9%]
	D+C+E+c+e+	0	0.0%	[0.0%, 3.7%]
Duffy	Fy(a-b-)	88	89.8%	[82.2%, 94.4%]
	Fy(a-b+)	5	5.1%	[2.2%, 11.4%]
	Fy(a+b-)	4	4.1%	[1.6%, 10.0%]
	Fy(a+b+)	1	1.0%	[0.2%, 5.6%]
Kidd	Jk(a+b-)	63	64.3%	[54.4%, 73.1%]
	Jk(a+b+)	31	31.6%	[23.1%, 41.5%]
	Jk(a-b-)	2	2.0%	[0.6%, 7.1%]
	Jk(a-b+)	2	2.0%	[0.6%, 7.1%]
Kell	K-k+	96	98.0%	[93.0%, 99.5%]
	K+k+	2	2.0%	[0.5%, 7.0%]

Bivariate and Inferential Analyses

Correlation between age and cumulative transfusions was not statistically significant (ρ = -0.034, 95% CI [-0.231, +0.166], p = .740).

Transfusion frequency did not differ significantly between females (Mean Rank: 50.56) and males (Mean Rank: 48.96; Mann-Whitney U = 908.50, p = .737).

Table 5:
Comparison of Transfusion Burden by Sex (Mann-Whitney U Test)

Sex	Count (n)	Mean Rank	Sum of Ranks	Test Statistic (U)	p-value
Female	26	50.56	1314.50	908.50	.737
Male	72	48.96	3536.50		

Tests of independence indicated no significant association between patient sex or municipality of origin and the expression of tested variable blood group antigens (p > .05).

Table 6:
Independence Testing of Antigen Frequencies Across Sex and Geographic Origin

Target Antigen	Sex Association (p-value)	Geographic Origin (p-value)	Conclusion
C	.673	.455	Independent
E	.228	.498	Independent
Kel-1 (K)	1.000	.703	Independent
Kel-2 (k)	1.000*	.608	Independent
Fy^a	.390	.546	Independent
Fy^b	.069	.593	Independent
Jka	.762	.515	Independent
Jk^b	.469	.742	Independent
M	.644	.292	Independent
N	1.000	.218	Independent
S	.929	.620	Independent

**Fisher's exact test applied due to small cell counts.*

DISCUSSION

Extended immunohematological mapping in this pediatric cohort provides baseline data on red cell antigen distribution among SCD patients in Lubumbashi, DRC.

An unexpected finding was the absence of a correlation between age and cumulative transfusion count (ρ = -0.034, p = .740). While cumulative transfusion exposure typically increases with age, severe acute complications—such as hyperhemolytic crises, splenic sequestration, and severe malarial anemia—occur frequently in early childhood in sub-Saharan Africa, concentrating transfusion events in younger age brackets (Novelli & Gladwin, 2016). Survival bias may also contribute to this pattern, as children with severe SCD phenotypes face higher early mortality in resource-limited settings (Katamea, 2024).

The ABO blood group system is marked by a clear predominance of Group O, representing 57.14% of subjects. This result is consistent with epidemiological data classically observed in many populations, particularly in sub-Saharan Africa, where Group O is predominant (Tagny et al., 2010). Blood group B is the second most common (30.61%), group A is relatively rare (11.22%), and group AB is almost nonexistent in this sample (1.02%), highlighting its usual rarity.

The detection of Duffy active antigens (Fy^a: 5.1%; Fy^b: 6.1%) and the Fy(a+b+) phenotype (1.0%) contrasts with literature describing Central African populations as near-homozygous for Duffy-null alleles driven by *Plasmodium vivax* selection pressure (Howes et al., 2011). This pattern suggests genetic admixture within urban Lubumbashi.

High transfusion rates among young subjects (40.8% with ≥ 3 transfusions) may be exacerbated by *Plasmodium falciparum* malaria, which causes acute anemia requiring emergency transfusion. Variable blood ordering practices and clinical transfusion thresholds across facilities may also contribute to high utilization rates.

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The male predominance in our sample (73.5%) did not correspond to a difference in transfusion burden compared to females (p = .737). This asymmetry likely reflects non-probabilistic convenience sampling and local healthcare-seeking patterns rather than biological sex differences in disease severity.

Current protocols under the National Blood Transfusion Program (PNTS) of the DRC limit pre-transfusion testing to ABO and RH1 (D) matching. Extended Rhesus antigens (C, c, E, e) and minor systems (KEL, FY, JK, MNS) are not routinely typed. Given that 88.8% of patients are C-negative and 92.9% are E-negative, transfusing unscreened blood presents a risk of primary alloimmunization to C and E antigens.

High rates of antigen-negative profiles, including Fy^a(-b-) (89.8%) and Jk(b-) (66.3%), underscore the potential value of targeted donor screening. Unmonitored antigen mismatches remain a primary driver of red cell alloimmunization in polytransfused patients (Chou et al., 2020; Pirenne et al., 2023).

Policy and Clinical Implications

i. **Transition to Extended Phenotyping:** The PNTS should consider expanding pre-transfusion typing protocols for multi-transfused pediatric patients to include extended Rhesus (C, c, E, e) and KEL1 antigens.

ii. **Donor Registries:** Establishing registries of phenotyped blood donors—including family-directed donor screening—may assist in securing antigen-matched units for polytransfused patients.

Study Limitations

This study has several limitations. The sample size (N = 98) and non-probabilistic convenience sampling from a single urban center limit generalization to the broader population of Haut-Katanga Province or the DRC. Serological hemagglutination testing was used exclusively, which cannot identify partial or weak antigen variants, silent alleles, or variant RH structures common in populations of African descent. Furthermore, because of the cross-sectional design, longitudinal monitoring of alloantibody development was not performed.

CONCLUSION

This study established an extended erythrocyte phenotype map for a pediatric SCD cohort in Lubumbashi, DRC. The findings demonstrate complete invariance for D, c, e, and s antigens alongside variations in minor blood group antigens, notably C (11.2%), E (7.1%), and a high prevalence of the Duffy-null profile Fy(a-b-) (89.8%). These results highlight immunogenetic risks under current ABO-RhD-only transfusion matching protocols. Adopting extended Rhesus and Kell matching strategies alongside phenotyped donor registries could improve transfusion safety for pediatric SCD patients in the region. Biologically, while stable invariants were confirmed (100% prevalence for D, c, e, and s) and a high prevalence of blood group O, significant phenotypic variations were highlighted in critical minor systems, particularly for the C (11.2%) and E (7.1%) antigens, as well as a predominant Duffy-null Fy(a-b-) phenotype (89.8%). These specific variations reveal a substantial, latent risk for anti-erythrocyte alloimmunization within the local healthcare framework.

Use of Artificial Intelligence (AI) Declaration: No artificial intelligence tools were used in the generation, primary analysis, or interpretation of the experimental raw data presented in this study.

Authors' Contributions: FA: Conceptualization, methodology, formal analysis, writing – original draft, supervision, validation. MV: Investigation, methodology, formal analysis, writing – review & editing. JPK-T-N: Conceptualization, literature review, validation, writing – review & editing. All authors read and approved the final manuscript.

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