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Extended Erythrocyte Phenotypical Profile in Patients with Sickle Cell Disease: Literature Review and Implications for Transfusion Safety

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ABSTRACT

Introduction
Erythrocyte alloimmunization remains a profound and pervasive complication in the clinical management of sickle cell disease (SCD).

Purpose
This study evaluates the global pooled prevalence of red blood cell antibodies and structural minor blood group antigenic variations that compromise transfusion safety across international cohorts.

Methods
Following PRISMA guidelines, a systematic literature search was performed using PubMed and Google Scholar. Nine high-quality observational studies published between 2012 and 2025 were selected, aggregating data from 1,241 multi-transfused SCD patients across four continents. To account for baseline clinical and geographical diversity, raw proportions were normalized via logit transformation and synthesized using a random-effects model.

Results
The global pooled prevalence of erythrocyte alloimmunization among multi-transfused SCD patients was 17.45% (95% CI: [10.09, 26.67]). Extreme statistical heterogeneity was observed (I^2 = 91.32%, Q = 92.146, p < 0.001), highlighting critical discrepancies between advanced proactive matching facilities (8.05%) and heavily pre-sensitized cohorts (68.42%). Phenotypic risk analysis revealed a 72.72% overall prevalence of the evolutionary null Duffy phenotype Fy(a-b-), a low KEL1 antigen prevalence of 3.32%, and an at-risk Kidd profile rate of 46.57%.

Conclusion
The high 17.45% alloimmunization rate reflects chronic antigenic exposure combined with systematic donor-recipient demographic mismatches. The evolutionary Fy(a-b-)} malaria-resistant profile acts as a major post-transfusion risk barrier when patients receive blood from dominant Caucasian donor pools. Transitioning from basic ABO-RhD compatibility to routine, extended serological phenotyping or molecular genotyping for minor systems (Rh subgroups, Kell, Duffy, Kidd, MNS) is a critical public health priority to eliminate delayed hemolytic transfusion reactions and optimize clinical care. **Systematic Review Registration:** PROSPERO CRD420261363920.

INTRODUCTION

Sickle cell disease (SCD) is the most widespread genetic blood disorder in sub-Saharan Africa and represents a major global public health challenge (Chou et al., 2020). The study of erythrocyte phenotypes in sickle cell patients is crucial where SCD is highly prevalent and imposes a heavy burden on healthcare systems (Özpolat et al., 2022). Red blood cell (RBC) alloimmunization is a common transfusion-related complication in these individuals, especially as life expectancy increases and clinical indications for chronic transfusion therapy expand (Pirenne et al., 2023). Human blood group polymorphism is defined by over 40 distinct blood group systems (Pirenne et al., 2023). Several minor systems require explicit attention in transfusion medicine because foreign antigens can induce alloantibody formation, precipitating immunohematological incompatibilities between donors and recipients (Pirenne et al., 2023).

Patients with SCD face heightened risks of severe clinical events, particularly acute vaso-occlusive crises (Novelli & Gladwin, 2016). Specific RBC antigenic phenotypes can modulate disease severity and the efficacy of therapeutic responses (Özpolat et al., 2022). Furthermore, frequent transfusion requirements necessitate precise determination of extended erythrocyte profiles to prevent adverse transfusion reactions (Chou et al., 2020). Characterizing these patient populations yields tailored data within specific geographic and genetic contexts (Pathare & Alkindi, 2017). This detailed mapping of phenotypic frequencies facilitates the identification of high-risk antigen profiles and allows for accurate risk stratification (Pathare & Alkindi, 2017).

Transfusion therapy remains a cornerstone of SCD management by lowering the fraction of sickle hemoglobin ({HbS}) to mitigate or prevent severe vaso-occlusive and ischemic complications (Gehrie et al., 2021). Whether administered via simple transfusion or automated exchange, RBC therapy occurs in both acute emergency settings and long-term prophylactic protocols (Gehrie et al., 2021). However, in many developing nations, multi-transfused patients do not routinely benefit from extended phenotypic matching across minor erythrocyte systems, resulting in high rates of alloimmunization (da Cunha Gomes et al., 2019). Alloimmunization—the formation of recipient antibodies against foreign donor antigens—is driven by multiple factors, including total transfusion exposure, underlying recipient neuro-inflammatory status, and recipient-donor antigenic disparities (Bernit et al., 2023; Chinedu et al., 2025; Martins et al., 2022). Limited routine data regarding minor blood group systems (Duffy, Kidd, MNS) exacerbates this problem, rendering unmonitored transfusions hazardous (Oladipupo et al., 2024).

METHODS

Protocol Registration and Search Strategy

The protocol for this systematic review was registered a priori in the International Prospective Register of Systematic Reviews (PROSPERO) under registration number CRD420261363920. The review was conducted and reported in strict accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.

A systematic literature search was conducted across PubMed and Google Scholar to identify biomedical studies and clinical research focusing on SCD, extended RBC phenotyping, and immunohematological outcomes. Predefined eligibility criteria based on population, exposure, and outcome variables were applied. Search strategies utilized combined keywords in English and French:

1. (Sickle Cell Disease OR Sickle Cell Anemia) AND (Extended Phenotyping OR Extended Match) AND (Alloimmunization OR Allo-immunization)
2. (Sickle Cell Disease) AND (Rhesus OR Kell OR Duffy OR Kidd OR MNS) AND (Transfusion Safety)

The search targeted primary observational studies published between 2012 and 2025 to reflect technological advances in immunohematology, such as high-throughput molecular genotyping. Literature filtering adhered strictly to PRISMA guidelines.

Inclusion Criteria

a) Primary observational studies focusing specifically on erythrocyte phenotypic distribution in SCD populations.

b) Cohorts reporting quantitative outcomes on alloimmunization and extended antigen matching
c) Studies published between 2012 and 2025 providing clear denominators and numerators.

Exclusion Criteria

a) Studies focusing on non-sickle cell chronic hemolytic conditions.

b) Studies lacking clear quantitative methodology or missing raw event counts.

c) Narrative reviews, institutional guidelines, editorial comments, and single-patient case reports (N = 1).

Study Selection and Data Management

Bibliographic citations were imported into the Rayyan systematic review platform. Two independent reviewers conducted initial blind screening of titles and abstracts. Blinding was unblinded only after initial selection to resolve discrepancies. Full-text articles were evaluated against eligibility criteria.

The selection process is detailed in the PRISMA Flow Diagram **(Figure 1)**:

I. **Identification:** N = 63 unique records identified across databases (0 duplicates found).

II. **Screening:** N = 63 titles/abstracts screened; n = 26 excluded as non-relevant or non-target population.

III. **Eligibility:** N = 37 full-text reports retrieved and assessed (0 retrieval failures). Excluded n = 28 reports due to:

(ABO, Rh subgroups, Kell, Duffy, Kidd, MNS).

i. Lack of quantitative numerator/denominator data (n = 24)

ii. Narrative review design (n = 2: Linder & Chou, 2021; Pirenne et al., 2023)

iii. Institutional practice guidelines (n = 1: ASH 2020)

iv. Isolated case report (n = 1: Wang et al., 2024)

IV. **Inclusion:** N = 9 primary studies met all criteria and were included in the quantitative meta-analysis.

Methodological Quality and Risk of Bias Assessment

The methodological quality of included observational studies was assessed independently by two reviewers using the Newcastle-Ottawa Scale (NOS) adapted for cross-sectional and cohort studies **(Table 1)**. Assessment evaluated three core domains:

A. **Selection** (Maximum 4 stars): Sample representativeness, size justification, and validated diagnosis of SCD.

B. **Comparability** (Maximum 2 stars): Control for clinical confounders (e.g., cumulative transfusion volume, ethnic matching of donor pool).

C. **Outcome** (Maximum 3 stars): Verification of alloantibodies using validated serological testing (column agglutination, solid-phase) or molecular genotyping.

Figure 1:
PRISMA Flow Diagram of Study Selection.

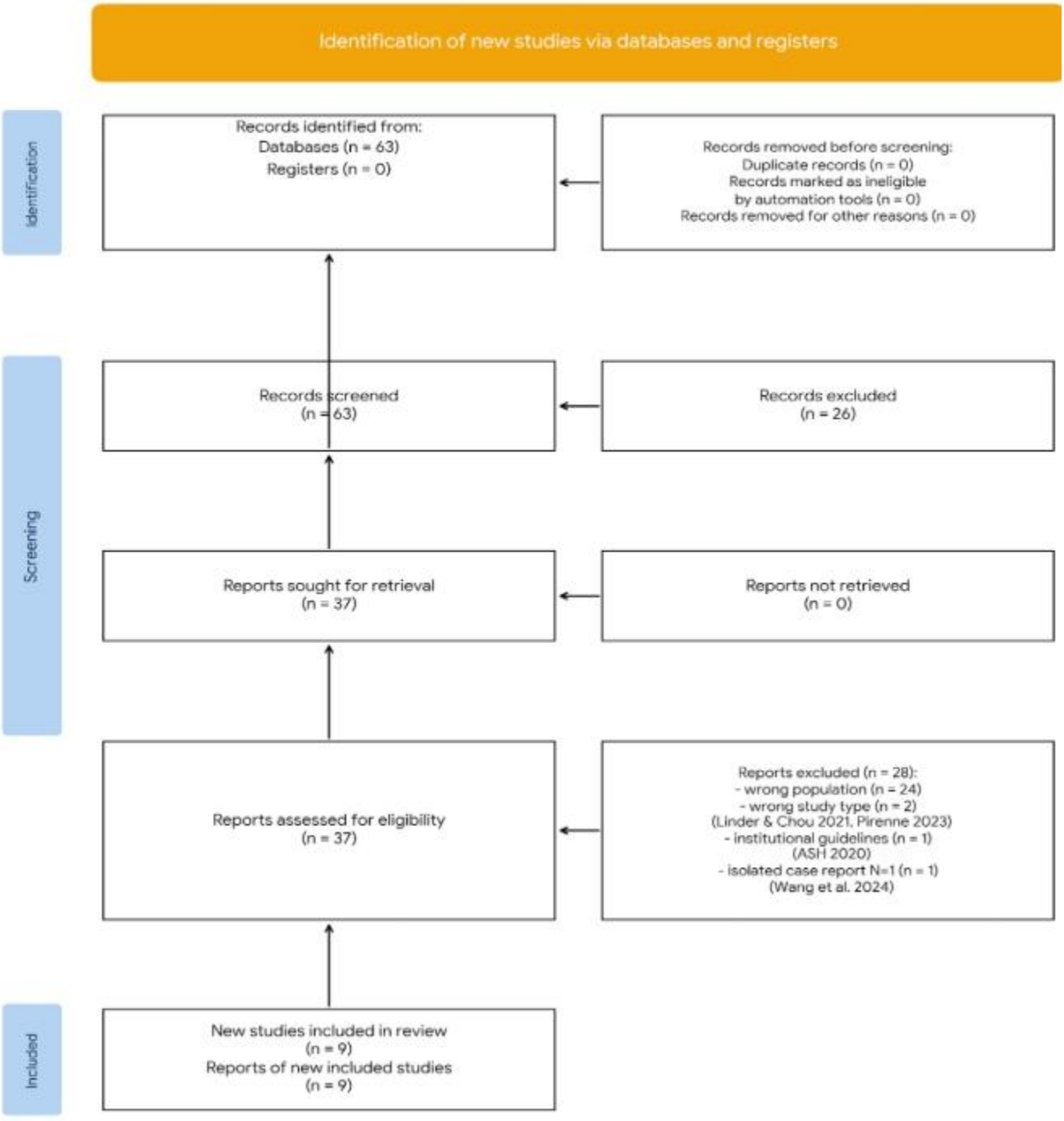

Table 1:
Methodological Quality Assessment of Included Studies (Newcastle-Ottawa Scale)

Primary Study (Year)	Selection (★)	Comparability (★)	Outcome / Exposure (★)	Cumulative Score	Quality Category
Felimban (2021)	★★★★	★☆	★★☆	7 / 9	**High**
Seck (2022)	★★★★	★★	★★☆	8 / 9	**High**
Boateng (2019)	★★★★	★☆	★★★	8 / 9	**High**
Chinedu (2025)	★★★★	★★	★★☆	8 / 9	**High**
Breiki (2024)	★★★☆	☆☆	★★☆	5 / 9	**Moderate**

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Leal (2023)	★★★★	★☆	★★☆	7 / 9	**High**
Conrath (2021)	★★★★	★★	★★★	9 / 9	**High**
Alkindi (2017)	★★★★	★☆	★★☆	7 / 9	**High**
Regalado-Artamendi (2021)	★★★★	★★	★★★	9 / 9	**High**

Statistical Analysis and Meta-Analysis Model

To compute pooled proportions without boundary distortion ($0 \leq p \leq 1$), raw study event rates were transformed using a Logit Transformation:

$$\text{logit}(p) = \ln(p/1 - p)$$

Variance of the logit-transformed proportion was calculated as:

$$SE(\text{logit}(p))^2 = 1/Np(1 - p)$$

Because included cohorts exhibited significant clinical and geographical diversity, meta-analytic pooling was performed using a Random-Effects Model (DerSimonian and Laird approach).

Heterogeneity was evaluated using:

a. **Cochran's Q test:** Evaluated at $\alpha = 0.05$.

b. **Inconsistency Index (I^2):** $I^2 = (Q - df)/Q \times 100\%$, where values >75% denote extreme heterogeneity.

c. **95% Prediction Interval (PI):** Calculated to forecast the expected range of true effects in future clinical settings.

All statistical calculations were executed using Comprehensive Meta-Analysis (CMA) Version 4.0.

RESULTS

Descriptive Characteristics of Included Studies

Nine primary studies met all eligibility criteria, capturing a total sample size of 1,241 multi-transfused SCD patients across eight countries (Saudi Arabia, Senegal, Ghana, Nigeria, Oman, Brazil, French Guiana, Spain). Over 75% of included data were published between 2021 and 2025.

Table 2:
Baseline Characteristics of Included Primary Studies

Study	Country	Patient Population / Genotypes	Sample Size (N)	Alloimmunized Cases (n)	Unadjusted Prevalence (%)	Dominant Antigen Systems & Specificities Identified
Felimban et al. (2021)	Saudi Arabia	SCA & beta-Thalassemia (Pediatric & Adult)	137	9	6.57%	Rh (C, c, E, e), Kell (K). Predominant: Anti-K
Seck et al. (2022)	Senegal	Homozygous SCD (HbSS = 95.3%)	153	24	15.69%	Rh, Kell. Dominant: Anti-Rh (34.2%), Anti-K (23.7%)
Boateng et al. (2019)	Ghana	Poly-transfused SCD (Median age: 9 yrs)	154	10	6.49%	Rh, Kell, Duffy, Kidd, MN. Identified RHCE variants
Chinedu et al. (2025)	Nigeria	Multi-transfused SCD (Abuja cohort)	148	26	17.57%	ABO, Extended Rh, Kell, Duffy, Kidd
Al Breiki et al. (2024)	Oman	Sickle Cell Disease (Extended typing cohort)	38	26	68.42%	Extended Rh, Kell, Duffy, Kidd (Highly sensitized)
Leal et al. (2023)	Brazil	SCD under prophylactic antigen matching	95	12	12.63%	Evaluated prophylactic matching efficacy
Conrath et al. (2021)	French Guiana	Poly-transfused SCD (Migratory population)	296	68	22.97%	Inter-ethnic donor-recipient disparities
Alkindi et al. (2017)	Oman	SCD & Thalassemia mixed cohort	133	42	31.58%	Single-center registry tracking long-term outcomes
Regalado-Artamendi et al. (2021)	Spain	SCD cohort at Tertiary Reference Center	87	7	8.05%	Complete Rh-Kell proactive matching model
TOTAL / SYNTHESIS	**Global**	**Severe Sickle Cell Syndromes**	**1,241**	**224**	**17.45%**	**Random-Effects Pooled Estimate**

Global Pooled Prevalence of Erythrocyte Alloimmunization

The pooled prevalence of erythrocyte alloimmunization was calculated using a random-effects model based on the logit-transformed proportions.

Table 3:
Meta-Analysis Event Rates and 95% Confidence Intervals Across Included Studies

Primary Study	Country	Events (n)	Total (N)	Event Rate (p)	95% Confidence Interval	Relative Weight (%)
Felimban et al. (2021)	Saudi Arabia	9	137	0.066	[0.035, 0.121]	10.74%

Seck et al. (2022)	Senegal	24	153	0.157	[0.107, 0.223]	12.43%
Boateng et al. (2019)	Ghana	10	154	0.065	[0.035, 0.116]	10.98%
Chinedu et al. (2025)	Nigeria	26	148	0.176	[0.122, 0.246]	12.56%
Al Breiki et al. (2024)	Oman	26	38	0.684	[0.522, 0.811]	9.42%
Leal et al. (2023)	Brazil	12	95	0.126	[0.073, 0.209]	11.21%
Conrath et al. (2021)	French Guiana	68	296	0.230	[0.185, 0.281]	13.88%
Alkindi et al. (2017)	Oman	42	133	0.316	[0.243, 0.399]	12.82%
Regalado-Artamendi et al. (2021)	Spain	7	87	0.081	[0.039, 0.159]	9.96%
Pooled Random-Effects Model	**Global**	**224**	**1,241**	**0.1745**	**[0.1009, 0.2667]**	**100.00%**

Summary Heterogeneity Statistics

i. **Cochran's Q:** 92.146 (df = 8, p < 0.001)

ii. **Inconsistency Index (I^2):** 91.32%

iii. **95% Prediction Interval:** [0.029, 0.602]

The global pooled prevalence of erythrocyte alloimmunization was 17.45% (95% CI: [10.09%, 26.67%]). Substantial statistical heterogeneity was observed (I^2 = 91.32%), reflecting variations in regional matching protocols and baseline transfusion burdens across cohorts.

Synthesis of Specific Antigen and Phenotypic Frequencies

Analysis of minor blood group systems demonstrated distinct phenotypic patterns in SCD patients.

Table 4:
Pooled Phenotypic Frequencies for Key Minor Antigen Systems

Blood Group System	Analyzed Phenotype / Antigen Profile	Number of Cohorts	Pooled Random-Effects Rate	95% Confidence Interval	Heterogeneity (I2)
Kell	KEL1 (K) Antigen Positivity	2	3.32%	[1.44%, 7.43%]	0.00%
Duffy	Fy(a-b-) Null Phenotype	2	72.72%	[55.25%, 85.20%]	71.90%
Kidd	At-Risk Kidd Phenotypic Profiles	2	46.57%	[4.83%, 93.74%]	99.29%
ABO	Blood Group O Prevalence	11	47.71%	[45.95%, 49.47%]	0.00%

DISCUSSION

Erythrocyte alloimmunization results from an immune response triggered by donor red cell antigens—primarily membrane proteins and glycoproteins—leading to alloantibody production and immunological memory (Jeyamurugan et al., 2024). Individual risk varies based on recipient age, total transfusions, underlying inflammatory state, and structural antigenic differences between recipients and donor pools (Dinardo et al., 2021). Alloimmunization restricts the availability of compatible RBC units and increases the risk of severe delayed hemolytic transfusion reactions (DHTR) and hyperhemolysis syndrome (Compernolle et al., 2018).

This meta-analysis establishes a global pooled erythrocyte alloimmunization prevalence of 17.45% in multi-transfused SCD patients. This rate is substantially higher than the 1–3% observed in general transfusion populations, driven by chronic exposure to non-self antigens and the pro-inflammatory environment of SCD (Chou et al., 2020; Pirenne et al., 2023). These findings align with broader literature; da Cunha Gomes et al. (2019) reported regional rates ranging from 4% to 47%, and Linder and Chou (2021) noted up to a tenfold higher risk in SCD compared to other chronically transfused conditions.

The pronounced heterogeneity (I^2 = 91.32%) highlights differences in international transfusion strategies. Lower alloimmunization rates in Spain (8.05%) and Saudi Arabia (6.57%) correlate with systematic, proactive extended Rh-Kell matching (Felimban et al., 2021; Regalado-Artamendi et al., 2021). Conversely, higher rates observed in certain Omani cohorts (31.58%–68.42%) reflect high transfusion burdens prior to the implementation of extended matching protocols (Alkindi et al., 2017; Al Breiki et al., 2024).

Antigenic Disparities and Clinical Implications

a) **Duffy System:** The high prevalence of the null Fy(a-b-) phenotype (72.72%) provides evolutionary resistance against *Plasmodium vivax* malaria. However, it presents a significant barrier in transfusion medicine when patients receive blood from predominantly Caucasian donor pools where Fy^a and

Fy^b are common (Jeyamurugan et al., 2024; Oladipupo et al., 2024).

b) **Kell System:** The low prevalence of the KEL1 (K) antigen (3.32%) underscores its immunogenic importance. Because KEL1 is highly immunogenic, even limited exposure can induce anti-K formation, a leading cause of severe DHTRs and obstetric complications (Pincez et al., 2022; Pirenne et al., 2023).

c) **Kidd System:** At-risk Kidd profiles (46.57%) present diagnostic challenges due to the tendency of anti-Jk^a and anti-Jk^b titers to fall below detectable levels over time, increasing the risk of delayed hemolytic reactions upon re-exposure (Linder & Chou, 2021; Shih et al., 2020).

Recommendations and Limitations

These results support guidelines from the American Society of Hematology recommending extended phenotypic matching (Rh, Kell, Duffy, Kidd) from the onset of transfusion therapy in SCD (Chou et al., 2020). In resource-limited settings where serological reagents are scarce, high-throughput molecular genotyping offers a scalable alternative to identify complex variants, particularly within the highly polymorphic Rh system (RHD/RHCE) (Karafin & Howard, 2022).

Limitations

1. The number of studies meeting inclusion criteria for specific Duffy and Kidd quantitative analyses was relatively small.
2. Inter-study methodological variations in antibody detection techniques contributed to statistical heterogeneity.
3. Epidemiological data from Central Africa remain underrepresented despite the region's high SCD burden.

CONCLUSION

This systematic review and meta-analysis demonstrates that erythrocyte alloimmunization remains a significant challenge in sickle cell disease, with a global pooled prevalence of 17.45%. This burden is driven by chronic transfusion requirements and antigenic mismatches between donor pools and recipient populations. The high prevalence of the Fy(a-b-) phenotype (72.72%) illustrates how an evolutionary adaptation to malaria increases post-transfusion risk when donor matching is limited to basic ABO-RhD compatibility.

Improving transfusion safety requires transitioning to routine extended phenotyping or molecular genotyping for minor systems (Rh subgroups, Kell, Duffy, Kidd, MNS). Expanding diagnostic infrastructure in high-burden, resource-limited regions is essential to prevent delayed hemolytic transfusion reactions and optimize long-term outcomes for patients with sickle cell disease globally.

Use of Artificial Intelligence (AI) Declaration: Bibliographic citations were imported and managed using the Rayyan systematic review platform. RAYYAN IS an AI TOOL.

Authors' Contributions:
O. A. F.: Conceptualization, Methodology, Software, Validation, Formal analysis, Investigation, Data Curation, Writing – original draft. **V. M.**: Validation, Supervision, Writing – review & editing. **K. T. N.**: Supervision, Writing – review & editing.

Data Availability Statement: Data supporting this meta-analysis were extracted from publicly available primary clinical studies cited within the text. Extracted datasets are available from the corresponding author upon reasonable request.

Ethical Approval: This literature review was conducted as part of a larger study that received ethical approval from the Bioethics Committee of the Higher Institute of Medical Techniques of Kinshasa (ISTM-Kinshasa) under Decision No. 055/ESU/ISTM/DG/2022 and Notice No. 183 CBE/ISTM/KIN/RDC/PM BBL/2025. However, because this specific manuscript utilizes only published secondary data, separate ethical approval was not required.

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