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Profile of Sickle Cell Disease Phenotypes among Patients Attending the National Centre for Sickle Cell Disease Control in N'Djamena, Chad

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Abstract Background. Sickle cell disease is an important inherited haemoglobin disorder in sub-Saharan Africa. Facility-based data on electrophoretic profiles in Chad remain limited. Objective. To describe the distribution of haemoglobin electrophoresis profiles and selected demographic and haematological findings among individuals tested at the National Centre for Sickle Cell Disease Control in N'Djamena. Methods. We reanalysed an existing cross-sectional dataset of 208 consecutive participants from the reported November 2023–November 2025 study period. Haemoglobin profiles were recorded by capillary electrophoresis and haematological measures by automated blood count. Data were summarised descriptively. The original clinical classifications and laboratory records remain subject to author verification. Results. Among 208 records, HbSS was recorded in 173 (83.2%), HbS/β^+-thalassaemia-compatible profile in 26 (12.5%), HbSE in four (1.9%), HbSC in one (0.5%) and HbAS in four (1.9%). Mean age among 194 records with numeric ages was 11.63 ± 8.12 years; mean haemoglobin was 6.23 ± 1.55 g/dL. Parental consanguinity was recorded in 116 (55.8%). Sex coding was unresolved in two records. Conclusion. The recorded electrophoretic profiles were predominantly HbSS. HbAS profiles must be reported as sickle cell trait rather than disease. The referral-center sample, potential effects of transfusion and absence of molecular confirmation limit genotype-level interpretation and generalizability.

Keywords: *sickle cell disease, capillary electrophoresis, haemoglobinopathy, HbSS, HbAS, Chad*

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1. Introduction

Sickle cell disease is a common inherited haemoglobin disorder and a major public-health challenge in sub-Saharan Africa [1,2,3,4] [5,6,7,8,9]. Accurate laboratory characterization is important for clinical management, genetic counselling and research.

Haemoglobin S results from a variant of the beta-globin chain. Polymerization of deoxygenated HbS contributes to haemolysis and vaso-occlusion, while the clinical presentation varies with haemoglobin genotype and disease modifiers [2,3,4,5].

HbSS, HbSC and HbS/β-thalassaemia can differ in laboratory and clinical characteristics. Distinguishing sickle cell disease phenotypes from HbAS (sickle cell trait) is essential for interpretation of results [3,4,5].

The distribution of sickling phenotypes differs across African populations [9,10,11,12] [13,14,15]. Hospital-based observations cannot, by themselves, establish population-level prevalence.

Capillary electrophoresis supports haemoglobin fraction identification, although recently transfused individuals and unusual or compound profiles may require additional diagnostic clarification. Molecular confirmation was not documented for the present cohort.

The objective of the present analysis was to describe the haemoglobin profiles recorded among all 208 tested participants, explicitly separating disease-associated sickling profiles from HbAS trait.

2. Materials and Methods

2.1. Study Design and Setting

A descriptive cross-sectional study was conducted at the National Centre for Sickle Cell Disease Control in N'Djamena, Chad. The centre provides specialised care, clinical follow-up, laboratory investigations, and management of patients with sickle cell disease and other haemoglobin disorders.

The study aimed to describe the distribution of haemoglobin phenotypes among patients attending the centre and to assess selected demographic and haematological characteristics according to the identified haemoglobin phenotypes.

The study was reported as taking place from November 2023 to November 2025; the exact start and end dates require confirmation against the study records.

2.2. Study Population and Sampling

The study population consisted of patients attending the National Centre for Sickle Cell Disease Control in N'Djamena during the study period who underwent haemoglobin phenotype testing.

Participants were recruited from patients attending the centre for consultation, routine follow-up, diagnosis, or management of sickle cell disease and other haematological disorders.

A total of 208 participants were included in the final analysis.

A non-probability consecutive sampling technique was used. All patients attending the National Centre for Sickle Cell Disease Control during the study period who fulfilled the predefined eligibility criteria were consecutively included in the study.

2.3. Laboratory Procedures

Venous blood samples were collected from eligible participants using standard laboratory procedures. Samples intended for haematological analysis were collected into tubes containing ethylenediaminetetraacetic acid (EDTA) as anticoagulant.

Blood samples were handled and processed according to the laboratory's standard operating procedures. The specimens were subsequently analysed for haemoglobin identification and quantification and for complete blood count parameters.

Haemoglobin phenotyping was performed using the MINICAP FLEX-PIERCING capillary electrophoresis system (Sebia, France). Capillary electrophoresis separates haemoglobin fractions according to their electrophoretic mobility and allows the detection and quantification of major haemoglobin fractions and clinically relevant haemoglobin variants. The electrophoretic profiles obtained were interpreted according to the laboratory's established criteria and classified into the corresponding haemoglobin phenotype categories. In the present study, the main disease-associated phenotypes identified were HbSS, HbS/β^+-thalassaemia, HbSC, and HbSE. HbAS profiles, when identified, were considered to represent sickle cell trait and were distinguished from disease-associated phenotypes.

A complete blood count (CBC) was performed using the Mindray BC-30 automated haematology analyser (Mindray, China). Venous blood samples collected in EDTA tubes were analysed according to the manufacturer's operating procedures and the laboratory's standard operating procedures.

The following haematological parameters were recorded: haemoglobin concentration (Hb); haematocrit (HCT); red blood cell count (RBC); mean corpuscular volume (MCV); mean corpuscular haemoglobin (MCH); mean corpuscular haemoglobin concentration (MCHC); red cell distribution width, coefficient of variation (RDW-CV); red cell distribution width, standard deviation (RDW-SD); white blood cell count (WBC); and platelet count (PLT). These parameters were used to describe the haematological characteristics of the study population and to assess their distribution across the identified haemoglobin phenotype groups where applicable.

2.4. Reanalysis of the Source Dataset

The study dataset was reported to contain 208 numerically identified records and 22 columns. Analysis used the raw category codes in the source spreadsheet. Profile codes SS, SB+, SE, SC and AS were retained as recorded; the code SB+ is described as compatible with HbS/β^+-thalassaemia, without implying genetic confirmation. Sex codes M and m were combined for descriptive tabulation; two entries labelled A+ were treated as invalid or undetermined sex, pending source review. No records were excluded solely because of an invalid sex code. Numeric means and sample standard deviations were recalculated from available data.

2.5. Ethical Considerations — author Confirmation Required

The source manuscript describes institutional research authorization and confidentiality safeguards. A signed authorization, documented consent/waiver determination and applicable institutional ethics documentation were not provided for independent review. These claims must be verified and finalized by the investigators before submission.

Table 1. Sex codes recorded in the original dataset

Original data code	Count	Interpretation
M/m	106	Male (standardised)
F	100	Female
A+	2	Sex undetermined
Total	208	

3. Results

3.1. Participants and Phenotype Distribution

The spreadsheet included 208 records. The recorded haemoglobin profiles are shown in Table 2. Four HbAS profiles (1.9%) were distinguished from the 204 records with disease-associated sickling profiles. These are dataset

classifications rather than molecularly confirmed genotypes.

Table 2. Electrophoretic profiles recorded among 208 participants

Electrophoretic profile	Count (n)	% of all 208 observations
HbSS	173	83.2
Compatible with HbS/β⁺-thalassaemia	26	12.5
HbSE	4	1.9
HbSC	1	0.5
HbAS (sickle cell trait)	4	1.9
Total	208	100.0

3.2. Demographic Characteristics

Among 194 age-valid records, age ranged from 1 to 37 years (mean 11.63 ± 8.12). The source sex column contained 104 M, 100 F, two lowercase m and two A+ codes. Case-standardization yields 106 male and 100 female records, with two undetermined entries; this coding interpretation requires source confirmation. Parental consanguinity was recorded in 116 participants (55.8%).

3.3. Haematological Characteristics

Table 3 presents descriptive statistics recalculated from the source workbook. The platelet unit remains to be confirmed before submission.

Table 3. Descriptive demographic and haematological characteristics

Variable	Valid n	Mean ± SD	Minimum	Maximum
Age (years)	194	11.63 ± 8.12	1	37
Haemoglobin (g/dL)	208	6.23 ± 1.55	2.4	9.4
Haematocrit (%)	208	17.54 ± 4.46	6.8	26.8
MCV (fL)	208	86.88 ± 8.95	56.6	111.9
MCH (pg)	208	31.77 ± 3.84	22.1	42
MCHC (g/dL)	208	36.15 ± 3.13	23.9	44.1
RDW-CV (%)	208	22.49 ± 5.55	10	43.1
RDW-SD (fL)	208	65.80 ± 14.09	11.4	108.8
White blood cells (per µL; unit presumed)	208	21,735.43 ± 16,475.04	1,600	157,800
Platelets (source scale; unit to confirm)	208	390.88 ± 179.80	28	1,027

3.4. Transfusion History and Diagnostic Limitations

Prior transfusion was recorded as “yes” for 140 (67.3%) and “no” for 68 (32.7%). The timing of transfusions in relation to electrophoresis was not available in the reviewed workbook; interpretation of variant fractions may therefore be affected.

4. Discussion

The predominant recorded pattern was HbSS, representing 173 of 208 profiles (83.2%). Compared with the original manuscript, source-data review found lower counts for HbSS and HbS/β⁺-thalassaemia and four HbAS profiles not identified in its primary phenotype table. This distinction is material because sickle cell trait should not be represented as sickle cell disease. The centre-based recruitment population must not be interpreted as representative of the wider Chadian population.

HbS/β⁺-thalassaemia-compatible patterns and the rare HbSE classification merit additional scrutiny of original haemoglobin fraction traces and clinical or molecular records [16]. Recent transfusion may interfere with electrophoretic interpretation. As no molecular confirmation was evidenced in the materials reviewed, the results should be understood as recorded electrophoretic phenotypes.

Parental consanguinity was commonly recorded; however, causal interpretation is not justified by this cross-sectional design. Prior chi-square significance claims require reconsideration because sparse phenotype subgroups violate conventional asymptotic assumptions. Ethnic grouping analyses should be repeated only after documented category mappings are fixed.

Table 4. Reported parental consanguinity by electrophoretic profile

Parental consanguinity	HbSS	HbS/β⁺	HbSC	HbSE	HbAS	Total
Yes	99	16	1	0	0	116
No	74	10	0	4	4	92
Total	173	26	1	4	4	208

Table 5. Reported community affiliation: transliterated source categories

Community affiliation (provisional transliteration)	Count (n)	Percentage (%)
Arab	84	40.4
Wadai	30	14.4
Bilala	12	5.8
Kanbou	10	4.8
Hadjar	8	3.8
Fellata	8	3.8
Massalat	6	2.9
Kouka	6	2.9
Gorane	6	2.9
Hausa	6	2.9
Baguirmi	4	1.9
Kotoko	4	1.9
Sara Ngambaye	4	1.9
Bornou (source label 1)	4	1.9
Bornou (source label 2)	4	1.9
Marba	2	1.0
Moudougou	2	1.0
Wadai Abcharib	2	1.0
Hadjar Bedjo	2	1.0
Toundjour	2	1.0
Dajo	2	1.0

Table 6. Reported previous transfusions

Previous transfusion	Count (n)	Percentage (%)
Yes	140	67.3
No	68	32.7

5. Limitations

The single-centre consecutive sample limits generalizability. Four trait profiles are included in the full phenotyping cohort. Two sex values are invalid or unresolved. Date of last transfusion, diagnostic electrophoresis traces, genetic confirmation and original ethical supporting documents were unavailable for independent inspection. The results are reported descriptively without new inferential significance claims.

6. Conclusion

Recorded HbSS profiles predominated among the 208 participants tested at the centre. HbAS was observed in four participants and should be reported separately as trait. These descriptive findings can inform future diagnostic research, provided that laboratory classifications, ethics documentation and the full reporting denominator are confirmed.

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Competing Interests

The authors declare that they have no competing interests.

Authors' Contributions

Mahamat Ahmat Abousskine: Conceptualization, study design, data collection, data analysis, interpretation of results, and manuscript writing. Ahmed Agabeldour: Supervision, scientific review, interpretation of findings, data analysis and critical revision of the manuscript. Alcherif Hamid Mahamat: Methodology, laboratory investigations, data interpretation, and critical revision of the manuscript. Millimi Adam Djibril: Data collection, statistical analysis, interpretation of results, and critical revision of the manuscript. Hissen Ali Habibu: Data collection, literature review, interpretation of results, and manuscript revision. Younous Ibrahim Younous: Data collection, laboratory investigations. All authors: Contributed to the final review and approval of the manuscript and agreed to be accountable for all aspects of the work.

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Data Availability

The data from this study are not publicly available due to ethical and confidentiality restrictions. They may be obtained from the corresponding author upon reasonable request, subject to the required ethical and institutional approvals.

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