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Original Research Article

Anthropometric, Clinical, and Laboratory Profile of Young Homozygous SS Sickle Cell Adults in Mbuji-Mayi, Democratic Republic of the Congo: A Case-Control Study

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Abstract: ***Background*:** In the Democratic Republic of the Congo (DRC), decentralized access to disease-modifying therapies for sickle cell anemia remains fragmented. This study characterizes the baseline anthropometric, clinical, and laboratory profiles of young homozygous SS adults followed in Mbuji-Mayi, a remote under-resourced area, to identify simple and accessible surrogate markers for clinical monitoring. ***Methods*:** A descriptive and analytical cross-sectional study was conducted from February to April 2026 in Mbuji-Mayi, DRC. We included 58 stable sickle cell patients (HbSS, aged 15–40 years) and 58 healthy age- and sex-matched controls. Multivariate logistic regression analysis was restricted to 56 homozygous SS patients with complete data to identify independent predictors associated with phenotypic severity, defined according to international standards as a clinical burden exceeding 3 vaso-occlusive crises (VOC) per year. ***Results*:** The overall mean age was 20.6 ± 4.5 years. HbSS patients exhibited severe chronic wasting compared to controls (median BMI: 17.8 vs. 19.6 kg/m²; $p < 0.001$), structural relative auscultatory hypotension, and baseline resting hypoxemia (mean SpO_2: 95.5% vs. 97.9%; $p = 0.002$). Somatic symptoms such as palpitations (60.34%) and dyspnea (41.40%) predominated significantly in sickle cell patients. The average annual clinical burden included 4.3 ± 3.5 VOC episodes and 1.1 ± 1.4 blood transfusions. The fully adjusted multivariate model demonstrated that resting peripheral capillary oxygen saturation (SpO_2) was the primary independent protective factor against severe clinical phenotypes (aOR = 0.75; 95% CI: 0.57–1.00; $p = 0.047$). Body Mass Index (BMI) displayed a major protective trend approaching statistical significance (aOR = 0.72; 95% CI: 0.51–1.00; $p = 0.053$). Age did not exert a significant independent influence (aOR = 1.17; $p = 0.063$), while hemoglobin levels and reticulocyte counts lost statistical significance ($p > 0.05$). ***Conclusion*:** Tissue hypoxemia at rest and nutritional depletion dictate the clinical expression of phenotypic severity among homozygous SS adults in remote low-resource areas. Non-invasive monitoring of resting SpO_2 and BMI provides simple, accessible, and high-utility surrogate markers to guide risk stratification and optimize supportive care in decentralized settings.

Keywords: Sickle Cell Disease, Homozygous SS, Peripheral Oxygen Saturation, Body Mass Index, Low-Resource Settings, Mbuji-Mayi.

1. INTRODUCTION

Sickle cell disease remains the world's leading monogenic disease in terms of prevalence and morbidity, representing a major and persistent public health challenge in sub-Saharan Africa [1, 2]. In the Democratic Republic of the Congo (DRC), data from pioneering neonatal screening programs conducted by Tshilolo and colleagues [3], corroborated macroscopically by recent modeling from the Global Burden of Disease study [4], indicate a high prevalence of the sickle cell trait, heavily

burdening public health indicators. While improvements in care now allow an increasing proportion of patients to reach adulthood [5, 6].

Nevertheless, the majority of the available scientific literature in the DRC stems from cohorts followed within major urban academic centers, such as Kinshasa or Lubumbashi [7]. The clinical, anthropometric, and evolutionary profile of sickle cell adults living in landlocked provinces or low-resource cities, such as Mbuji-Mayi in Kasaï-Oriental, remains poorly documented. In these isolated regions, local cultural beliefs [8] and socio-economic constraints significantly limit regular access to validated disease-modifying therapies such as hydroxyurea [9].

This study aims to characterize the baseline anthropometric, clinical, and laboratory profile of homozygous SS sickle cell adults followed in Mbuji-Mayi, compared to a local population of healthy controls, to identify simple clinical monitoring markers exploitable in decentralized care settings.

2. MATERIALS AND METHODS

2.1. Study Design and Setting

A descriptive and analytical cross-sectional study was conducted over a three-month period, from February to April 2026, in Mbuji-Mayi, the capital city of the Kasaï-Oriental province in the Democratic Republic of the Congo (DRC). The operational organization of this research relied on the healthcare infrastructure of the Mbuji-Mayi Pediatric Clinic and the Megumi Medical Center. Clinical evaluations, anthropometric measurements, and laboratory analyses were centralized within these two medical facilities.

2.2. Study Population and Sampling

The study enrolled male and female subjects aged 15 to 40 years. From an initial pool of 129 individuals assessed for eligibility, 13 were excluded: one subject was excluded due to thalassemia diagnosed by capillary electrophoresis via the Gazelle system, and 12 were excluded due to the lack of strict matching or clinical exclusion criteria. A consecutive sampling method combined with a strict 1:1 matching protocol Consecutive sampling combined with a 1:1 age- and sex-matching protocol based on age and sex allowed the constitution of two final groups of 58 subjects each:

- **The Sickle Cell Group (Cases):** Comprised 58 homozygous SS patients identified by the HemoTypeSC rapid test or on the basis of a certified prior laboratory report. The absence of a vaso-occlusive crisis (VOC) during the four weeks prior to enrollment was required to ensure clinical stability. Within this cohort, clinical phenotype severity was quantitatively defined according to international standards as an annual frequency strictly greater than 3 acute VOCs per year (> 3 VOC/year), an epidemiological threshold validated as a major indicator of morbidity and early mortality in adults [10].
- **The Control Group (Controls):** Comprised 58 non-sickle cell subjects (37 AA and 21 AS) identified by the HemoTypeSC test, who were healthy volunteers recruited from among patient companions and members of the local community.

Common exclusion criteria for both groups included pregnancy, lactation, and recent acute infections. Furthermore, to eliminate major confounding factors during the evaluation of hemodynamic profiles and baseline somatic functional symptoms (palpitations, dyspnea), subjects with confirmed hypertension, congenital heart disease, or chronic respiratory pathologies were systematically excluded, in accordance with the complication phenotypes described in the literature[11, 12]. This methodological choice of strictly excluding known structural cardiorespiratoy pathologies isolates the purely systemic, metabolic, and peripheral microvascular impairment specific to stable SS adults, as these organ pathologies are the focus of a separate echocardiographic exploration workflow.

2.3. Data Collection and Clinical Procedures

Weight and height were measured according to anthropometric standards to calculate the Body Mass Index (BMI = weight / height²). Waist circumference, hip circumference, waist-to-hip ratio (WHR), and neck circumference were measured using a flexible measuring tape.

Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured on the right arm using the classical auscultatory method with a manual cuff sphygmomanometer and a stethoscope to detect Korotkoff sounds. Measurements were performed after a 15-minute rest in a seated position, in accordance with the methodological standardization guidelines of the American Heart Association (AHA) for measuring human blood pressure[13]. Resting peripheral capillary oxygen saturation (SpO_2) was measured simultaneously using a standardized pulse oximeter positioned on the index finger, according to the technical recommendations of the American Thoracic Society (ATS) [14]. The presence of chronic somatic functional symptoms (dyspnea, palpitations) was documented using a standardized interview questionnaire to assess the systemic impact of chronic anemia and peripheral hypoxemia[11,12]. For the SS group, the annual history of VOCs and blood transfusions was retrieved from medical charts.

On the laboratory front, baseline hemoglobin levels were quantified manually using Sahli's acid-haematin colorimetric method [15]. The reticulocyte count was determined by direct visual counting under an optical microscope after standardized supravital staining with brilliant cresyl blue, expressed as a percentage of the total number of red blood cells, in accordance with

validated reference protocols for medical biology laboratories operating in resource-limited settings [15]. Urinalysis for proteinuria was performed by visual inspection using reactive urine dipsticks.

The protocol was formally approved by the Institutional Ethics Committee of the University of Mbuji-Mayi (No. 02/CEI/UM/ESU/2026), and written informed consent was obtained from each participant prior to enrollment, in accordance with the Declaration of Helsinki [16].

2.4. Statistical Analysis

Data processing and analysis were performed using Stata/IC software v.13.0. The normality of distributions was systematically evaluated using the Shapiro-Wilk test. Continuous variables are expressed as Mean ± Standard Deviation (SD) for Gaussian distributions, or as Median [Interquartile Range - IQR] for non-normal variables. Categorical variables are presented as frequencies and percentages.

Comparisons were performed using Student's t-test (or Welch's t-test) or the Wilcoxon-Mann-Whitney test for quantitative variables, and Pearson's Chi-square test or Fisher's exact test for qualitative variables.

Factors independently associated with phenotypic severity within the homozygous SS cohort were evaluated using a global multivariate logistic regression sub-model. The independent variables introduced simultaneously into the adjusted model were selected based on a univariate threshold of $p < 0.20$ and their peripheral cardiorespiratory clinical relevance. Due to missing data in specific covariates, two HbSS patients were excluded from this analysis, restricting the final multivariate logistic regression model to 56 subjects. The threshold for statistical significance was set at $p < 0.05$.

3. RESULTS

The overall mean age of our sample was 20.6 ± 4.5 years among sickle cell SS patients and 20.5 ± 4.5 years among controls ($p = 0.823$), with a strict proportion of 55.17% female subjects (32 women per group) [Table 1]. The anthropometric, hemodynamic, clinical, and laboratory status of the patients compared to the controls is detailed in Table 1 [Table 1].

Table 1: Baseline sociodemographic, anthropometric, clinical, and laboratory characteristics of the participants

Variables	Sickle Cell SS (n=58)	Controls (n=58)	*p*-value
Sociodemographics			
Age (years) [a]	20.6 ± 4.5	20.5 ± 4.5	0.823
Female sex, n (%)	32 (55.17)	32 (55.17)	> 0.999
Anthropometric			
Weight (kg) [b]	48.5 [43.0 – 54.0]	55.0 [51.0 – 62.0]	< 0.001
Height (m) [b]	1.62 [1.58 – 1.68]	1.68 [1.64 – 1.73]	< 0.001
BMI (kg/m²) [b]	17.8 [16.2 – 20.2]	19.6 [18.3 – 22.2]	< 0.001
Waist circumference (cm) [b]	71.0 [68.0 – 75.0]	72.0 [68.0 – 76.0]	0.589
Hip circumference (cm) [b]	83.0 [77.0 – 87.0]	92.0 [86.0 – 96.0]	< 0.001
Waist-to-Hip Ratio [b]	0.86 [0.82 – 0.89]	0.78 [0.74 – 0.81]	< 0.001
Neck circumference (cm) [b]	31.0 [30.0 – 33.0]	33.5 [31.0 – 35.0]	< 0.001
Clinical			
SBP (mm Hg) [a]	104.0 ± 12.2	110.4 ± 11.3	0.004
DBP (mm Hg) [b]	60.0 [60.0 – 70.0]	70.0 [60.0 – 70.0]	0.005
Respiratory rate (cpm) [a]	20.8 ± 5.6	20.1 ± 2.4	0.312
SpO_2 (%) [a]	95.5 ± 5.6	97.9 ± 1.4	0.002
Dyspnea, n (%)	24 (41.40)	9 (15.52)	0.007
Palpitations, n (%)	35 (60.34)	21 (36.21)	0.016
Laboratory			
Hemoglobin (g/dL) [b]	8.1 [7.6 – 9.0]	12.0 [11.0 – 13.0]	< 0.001
Reticulocyte count (%) [b]	1.8 [1.6 – 1.9]	1.0 [0.6 – 1.5]	< 0.001
Positive proteinuria, n (%)	2 (3.45)	1 (1.72)	0.559

Note: [a] *Data expressed as Mean ± Standard Deviation (SD);* [b] *Data expressed as Median [IQR].*

The anthropometric status of sickle cell SS adults objectives a significant deficit compared to control subjects, marked by a low weight and a lower median BMI (17.8 vs. 19.6 kg/m²; $p < 0.001$) [Table 1]. Clinical examination shows a significant decrease in systolic ($p = 0.004$) and diastolic ($p = 0.005$) blood pressures, as well as peripheral oxygen desaturation at 95.5% ($p = 0.002$) [Table 1]. Chronic functional symptoms such as palpitations (60.34%) and dyspnea (41.40%) largely predominate in patients [Table 1].

The annual profile of morbidity and laboratory activity specific to homozygous patients is presented in Table 2.

Table 2: Annual clinical and laboratory morbidity profile of homozygous sickle cell patients (n=58)

Clinical and laboratory variables	Values
Sickle cell phenotype (SS), n (%)	58 (100)
Vaso-occlusive crises per year [a]	4.3 ± 3.5
Blood transfusions per year [a]	1.1 ± 1.4
Baseline hemoglobin level (g/dL) [a]	8.4 ± 1.1
Reticulocyte count (%) [a]	1.8 ± 0.3
Positive proteinuria, n (%)	2 (3.45)

Note: [a] *Data expressed as Mean ± Standard Deviation (SD).*

Retrospective data collection highlights significant clinical instability with an average of 4.3 vaso-occlusive crises per year and a need for annual transfusion support [Table 2].

The multivariate logistic regression model, restricted to homozygous SS patients with complete data (n = 56), is presented in Table 3. Two patients were automatically excluded by the software due to incomplete clinical covariates. After mutual adjustment of endogenous confounding factors, only resting peripheral capillary oxygen saturation (SpO_2) emerges as an independent protective factor statistically associated with the phenotypic severity of the disease (aOR = 0.75; 95% CI: [0.57–1.00]; $p = 0.047$). This result indicates that a 1% increase in SpO_2 is correlated with a 25% reduction in the risk of experiencing a high annual burden of acute vaso-occlusive crises. Body Mass Index (BMI) displayed a strong protective trend approaching statistical significance (aOR = 0.72; $p = 0.053$). Hemodynamic blood pressure parameters, sex, as well as hemoglobin and reticulocyte levels show no significant independent association with the level of clinical severity ($p > 0.05$).

Table 3: Multivariate logistic regression analysis of independent factors associated with clinical severity in homozygous SS sickle cell patients (N=56)

Predictive variables	Adjusted Odds Ratio (aOR)	95% Confidence Interval [95% CI]	*p*-value
Age (years)	1.17	[0.99 – 1.38]	0.063
Sex (Male)	0.81	[0.19 – 3.53]	0.777
BMI (kg/m²)	0.72	[0.51 – 1.01]	0.053
SBP (mmHg)	1.01	[0.94 – 1.08]	0.792
DBP (mmHg)	1.01	[0.93 – 1.09]	0.901
SpO_2 (%)	0.75	[0.57 – 0.99]	0.047*
Hemoglobin (g/dL)	0.90	[0.34 – 2.38]	0.837
Reticulocyte count (%)	1.24	[0.04 – 35.48]	0.899

Note: * *Statistically significant value at the p < 0.05 threshold. The model mathematically excludes healthy controls to focus solely on the intrinsic determinants of severity within the homozygous SS cohort.*

4. DISCUSSION

The results documented in our study highlight the severity of the phenotypic expression of homozygous sickle cell disease in young adults living in a landlocked sub-Saharan environment [7–9]. The global anthropometric deficit observed in our SS patients, characterized by a significantly lower weight and a borderline median BMI of 17.8 kg/m², corroborates the findings of recent studies conducted in Central Africa [7-17]. This persistent nutritional deficit is physiologically explained by the drastic increase in basal energy requirements imposed by the metabolic hyperactivity of compensatory erythropoiesis face to intense chronic hemolysis, as well as by the permanent endothelial inflammatory state inherent to this pathology [17,18].

The high annual rate of vaso-occlusive crises (4.3 ± 3.5 episodes/year) associated with a need for regular transfusion support (1.1 transfusions/year) demonstrated in our sample highlights the exposure of the adult population of Mbuji-Mayi to deleterious active morbidity. This high frequency of acute events documented in our field, which fills the gap of specific epidemiological data for this landlocked province, is consistent with the major complication rates reported in urban university hospital centers in Kinshasa by Mikobi [7]. It also confirms, at the local level, the persistence of socio-economic obstacles limiting access to disease-modifying therapies in the DRC described by Mbiya Mukinayi [9]. This high frequency of acute events directly reflects the weakness of local prevention strategies, particularly the major difficulties in accessing modern modulating therapies such as hydroxyurea outside major urban axes [9].

The upstream clinical expression, objectified in our series by the marked predominance of palpitations (60.3%) and dyspnea (41.4%) in SS cases, reflects the profound systemic impact of chronic hemolytic anemia (median Hb at 8.1 g/dL) and relative tissue hypoxemia

evidenced by the lowering of SpO_2 to 95.5% [Table 1]. Mechanistically, these clinical abnormalities observed in our field stem from the pathophysiological cascade described by Gladwin [11], where chronic intravascular hemolysis is accompanied by a massive release of cell-free hemoglobin that depletes endothelial nitric oxide (NO). This mechanism alters peripheral vasoreactivity and accentures the global somatic circulatory dysfunction detailed by Gladwin [19], forcing the cardiorespiratory system into a chronic adaptation to preserve oxygen delivery face to the collapse of the oxygen-carrying capacity of hemoglobin [20]. This resting symptomatic burden thus directly reflects disruptions in arginine and nitric oxide metabolism involved in the genesis of secondary vasculopathies characteristic of the disease [18-22].

Finally, the multivariate logistic regression analysis, strictly restricted to homozygous SS patients (N=56), provides a crucial pathophysiological insight to optimize therapeutic targeting in decentralized care settings. While the pioneering work of Makani [23], emphasizes the continental urgency of structuring management face to heavy silent adult mortality, the mutual adjustment of our variables isolated within the single sample of patients reveals that resting peripheral capillary oxygen saturation (SpO_2) constitutes the main significant independent protective factor against the risk of clinical severity [Table 3]. These data suggest that the level of basal tissue hypoxemia strongly influences phenotypic instability and the frequency of vaso-occlusive events. Mechanistically, the nature of this association warrants nuance: a low SpO_2 can be perceived both as a reflection of chronic pulmonary dysfunction induced by erythrocytic pathology and as the direct determinant of the shift toward hemoglobin S polymerization under the effect of local hypoxia. This observation is consistent with the endothelial dysfunction and vasculopathy models described by Gladwin and Kato [11-18], where somatic microvascular occlusion phenomena appear closely linked to the peripheral oxygenation profile [12], independently of the crude anemia level or regeneration kinetics.

Furthermore, the Body Mass Index (BMI) displays a major clinical protective trend, sitting at the borderline of statistical significance (aOR = 0.72; p = 0.053) [Table 3]. The nature of this effect also requires nuance: a low BMI reflects the baseline metabolic wasting imposed by the hyperactivity of compensatory erythropoiesis and chronic endothelial inflammation [17], but it also influences patient vulnerability by lowering systemic resistance face to acute ischemic insults [6]. Age, on the other hand, shows a simple clinical trend toward phenotypic worsening, without our model demonstrating any significant independent statistical influence (aOR = 1.17; p = 0.063) [Table 3]. This lack of significance suggests that the burden of vaso-occlusive events in adults in Mbuji-Mayi is more heavily dictated by current peripheral oxygenation variables (SpO_2) than by the accumulated temporal facto [5]. These chronic visceral injuries settle silently at the somatic level and fully justify the implementation of targeted and joint monitoring of SpO_2 and nutrition within decentralized healthcare facilities, in order to prevent long-term phenotypic degradation of the sickle cell adult [5-24].

5. CONCLUSION

The homozygous SS sickle cell adult living in Mbuji-Mayi is clinically characterized by marked staturo-ponderal deficit, profound anemia, and a daily burden of disabling functional symptoms. In this resource-limited setting, peripheral capillary oxygen saturation (SpO2) emerges as the main simple, non-invasive, and independent clinical indicator associated with disease severity, while the Body Mass Index (BMI) represents a pivotal nutritional marker of clinical vulnerability. The systematic and combined integration of these two accessible tools in decentralized monitoring would optimize risk stratification and the targeting of nutritional and therapeutic supportive care for patients in the province, ultimately improving their overall somatic prognosis.

Transparency Statement

This study is part of a comprehensive, multidisciplinary translational research project conducted in Mbuji-Mayi, assessing the multisystemic impact of homozygous sickle cell adults in a landlocked environment. The authors declare in full transparency that the raw data from this protocol have been utilized for separate, complementary, and independent scientific publications. The targeted exploration of cardiac geometric, valvular, and hemodynamic function via standard and tissue Doppler echocardiography is the subject of separate manuscripts submitted for publication (left and right axes). The choice to exclude hypertensive subjects or those with known structural heart disease in the present manuscript responds precisely to this necessity for separation: this article focuses exclusively and independently on anthropometric characterization, the wasting-inflammation profile, and baseline peripheral clinical or laboratory biomarkers exploitable in decentralized care settings, without any textual or analytical overlap with the aforementioned echocardiographic works.

Regulatory Declarations:

Conflicts of Interest: The authors declare no financial, commercial, or academic conflicts of interest regarding this manuscript.

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Ethical Approval: The research protocol was formally approved by the Institutional Ethics Committee of the

University of Mbuji-Mayi (No. 02/CEI/UM/ESU/2026). Written informed consent was obtained from each participant prior to enrollment, in accordance with the Declaration of Helsinki.

Authors' Contributions: D.K.K. and G.T.D. designed the study, supervised the overall workflow, and drafted the manuscript. I.N.M., R.M.M., and H.K.K. performed the laboratory biological and hematological analyses. D.K.C. and A.M.D. performed the rigorous collection of clinical and anthropometric data. E.M.M. transcribed the data into the case report forms. D.T.M. and M.K.M. managed the recruitment and matching of participants. B.M.M. facilitated patient access at the Pediatric Clinic and participated in the overall supervision. A.C.M. and C.-R.A. provided academic supervision and critical revision of the manuscript. All authors have read and approved the final version of the manuscript.

List of Abbreviations

- **AA / AS / SS:** Hemoglobin phenotypes (Normal / Sickle cell trait / Homozygous sickle cell anemia)
- **aOR:** Adjusted Odds Ratio
- **AHA:** American Heart Association
- **ATS:** American Thoracic Society
- **BMI:** Body Mass Index
- **DBP:** Diastolic Blood Pressure
- **DRC:** Democratic Republic of the Congo
- **95% CI:** 95% Confidence Interval
- **SBP:** Systolic Blood Pressure
- **SD:** Standard Deviation
- **SpO_2:** Peripheral capillary oxygen saturation measured by pulse oximetry
- **VOC:** Vaso-Occlusive Crisis / Crises
- **WHR:** Waist-to-Hip Ratio

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