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Prevalence of Anemia in Sickle Cell Patients in Ugandan Hospitals: A Narrative Review

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ABSTRACT

Sickle cell disease (SCD), particularly sickle cell anemia (HbSS), is intrinsically a chronic hemolytic anemia, but the *severity* and *hospital burden* of anemia vary widely across patients and care settings. In Uganda, where SCD is common and malaria and undernutrition remain important co-morbid risks, anemia drives symptoms (fatigue, pallor), precipitates admissions, and increases transfusion demand. Using published Ugandan hospital/clinic evidence and global hemoglobin (Hb) diagnostic thresholds, this review summarizes: (i) how anemia is defined in SCD, (ii) facility-based indicators of anemia severity in Ugandan SCD patients, and (iii) key drivers and clinical/public-health implications. Ugandan clinic data from Mulago show that ~25–28% of children in routine follow-up may have Hb <7 g/dL at assessment (notably excluding Hb <5 g/dL), while Northern Uganda hospitalization data report pallor in ~20% at presentation and transfusion in ~51% of admissions, suggesting frequent clinically significant anemia among inpatients. These findings underscore the need for standardized anemia severity reporting in SCD, routine evaluation for reversible contributors (malaria, parvovirus B19, iron deficiency where appropriate, helminths, renal disease), and context-appropriate transfusion and hydroxyurea strategies.

Keywords: Sickle cell disease, sickle cell anemia, anemia prevalence, hemoglobin, transfusion, Uganda, Mulago Hospital, Gulu Regional Referral Hospital

INTRODUCTION

Sickle cell disease (SCD) is a major inherited hemoglobinopathy and one of the most important non-communicable genetic disorders in sub-Saharan Africa. It results from a point mutation in the β-globin gene that produces hemoglobin S (HbS), which polymerizes under deoxygenated conditions, leading to erythrocyte sickling, microvascular obstruction, chronic inflammation, and progressive end-organ damage [1]. In high-burden African settings, SCD remains a recognized public health priority because it contributes substantially to childhood morbidity and mortality through recurrent painful vaso-occlusive crises, severe anemia, acute chest syndrome, stroke, splenic dysfunction, and heightened susceptibility to infections. The risk profile is amplified by structural health-system constraints, delayed diagnosis, limited access to comprehensive SCD care, and the high prevalence of infectious diseases and nutritional deficiencies that can worsen clinical outcomes [2].

In Uganda, national surveillance and newborn screening-related data have demonstrated substantial geographic variation in SCD burden, implying that risk is not uniformly distributed across the country. This heterogeneity matters for service delivery: regions and facilities serving high-prevalence communities are likely to experience higher caseloads of SCD-related complications, greater demand for diagnostics and blood transfusion services, and more frequent admissions for crisis management [3]. Facility-specific evidence is therefore essential for planning: it helps determine the real-world scale of anemia-related admissions, the clinical severity profile seen at different levels of care, and the transfusion needs that drive blood bank utilization.

Anemia is central to SCD pathophysiology. Chronic hemolysis and markedly shortened red blood cell lifespan underpin a persistent baseline anemia that is often compounded by reticulocytosis, hyperbilirubinemia, and compensatory marrow expansion. However, the clinical impact of anemia in SCD extends beyond "low hemoglobin"

values. Worsening anemia is a frequent trigger for referral, admission, urgent evaluation, and blood transfusion, particularly when associated with tachycardia, dyspnea, fatigue, syncope, heart failure risk, or impaired oxygen delivery during intercurrent illness [4]. Several common comorbid and precipitating factors in Ugandan and similar contexts can aggravate anemia severity: malaria and other febrile illnesses, bacterial infections, helminth-related iron loss, poor dietary intake, micronutrient deficiencies (including folate), dehydration, and acute marrow suppression. In addition, SCD patients are vulnerable to specific, high-risk etiologies of sudden hemoglobin decline, including aplastic crisis (often associated with parvovirus B19), splenic sequestration, and hyperhemolytic episodes—each of which can rapidly progress to life-threatening anemia and shock if not recognized and treated promptly [5].

Yet, measuring and reporting "prevalence of anemia" among SCD patients can be conceptually tricky. Because most patients with homozygous sickle cell anemia (HbSS) are chronically anemic by general-population thresholds, a simple prevalence of anemia may be less informative for clinical decision-making and health planning. What is often more actionable is the prevalence and pattern of *moderate-to-severe anemia*, *acute drops from individual baseline hemoglobin*, and *transfusion-requiring anemia*, as these metrics more directly reflect clinical severity, risk of adverse outcomes, resource utilization, and quality-of-care needs [6]. In practice, defining anemia by severity categories (mild, moderate, severe), and distinguishing chronic baseline anemia from acute exacerbations can better guide triage, admission criteria, transfusion protocols, and follow-up planning. This becomes especially important in facilities where diagnostic capacity is limited, blood supplies can be constrained, and clinicians must prioritize patients most likely to benefit from urgent interventions [7].

Despite the central role of anemia in SCD morbidity and health-service utilization, there remains a gap in facility- and region-specific evidence describing the burden and clinical pattern of anemia among SCD patients in Uganda. Many clinical services still rely on generalized assumptions—either extrapolated from broader national estimates or from non-local literature—without robust, context-specific data on how frequently moderate-to-severe anemia occurs, how often patients present with acute hemoglobin declines, and the proportion that becomes transfusion-requiring [8]. This gap limits effective planning for essential inputs (laboratory testing, triage systems, inpatient bed allocation, transfusion services, and follow-up care). It also constrains the ability to improve clinical outcomes through early recognition and targeted prevention of precipitants such as infections, nutritional deficits, and marrow suppression.

Additionally, when anemia prevalence is reported without stratifying by severity, acute-versus-chronic presentation, or transfusion requirement, it can dilute the clinical relevance of findings and reduce their utility for decision-making. In a setting where blood is a limited and life-saving resource, the absence of detailed, locally generated data on transfusion-requiring anemia among SCD patients risks both under-preparedness (leading to delays and poor outcomes) and inefficient allocation (overestimating demand without appropriate severity profiling) [9]. Therefore, there is a pressing need to generate evidence that is both clinically meaningful and operationally useful for SCD care in Uganda, particularly evidence that captures anemia severity patterns and care implications at the facility level. This study will determine the prevalence and clinical pattern of anemia among patients with sickle cell disease (SCD) managed at the study facility by quantifying the proportion with moderate-to-severe anemia, documenting episodes of acute hemoglobin decline from baseline where prior values exist, and estimating the share with transfusion-requiring anemia alongside their presenting features. These data are important for patients, clinicians, managers, and policymakers because they support sharper triage and earlier recognition of high-risk presentations, improve transfusion planning and blood-bank preparedness through more accurate demand forecasting, and enable better targeting of preventable triggers such as infections, malaria (where relevant), and nutritional deficiencies via folate/nutrition support and patient education. In addition, defining the local severity profile can guide staffing, laboratory resourcing, and follow-up systems for recurrent severe anemia, contribute facility-level evidence to Uganda's national SCD response, and highlight documentation gaps that can drive quality improvement and future research on predictors and outcomes.

Approach and scope

This narrative review synthesizes evidence from Ugandan hospitals and clinics on anemia profiling in sickle cell disease (SCD), focusing on studies and routine-care datasets that report hemoglobin (Hb) strata, anemia-related clinical features, and/or transfusion-related indicators (e.g., transfusion history, trigger thresholds, documented indications). The scope prioritizes Ugandan, patient-level or cohort-level reporting that enables interpretation of anemia severity patterns within real-world SCD care, including how Hb distributions align with symptom burden such as fatigue, dyspnea, palpitations, dizziness, poor exercise tolerance, jaundice, and growth or pregnancy-related complications where applicable. To enhance interpretability and comparability, findings are contextualized against contemporary global Hb thresholds used to define and grade anemia across populations (by age, sex, and pregnancy status). The review therefore integrates locally generated clinical evidence with internationally used cutoffs to clarify how anemia is characterized, recognized, and acted upon in Ugandan SCD settings, and to highlight gaps in reporting, thresholds, and transfusion decision-making that may affect care.

Defining anemia in SCD: why "prevalence" needs qualifiers

Defining anemia in sickle cell disease (SCD) requires careful qualification because "prevalence" depends heavily on the threshold used and the clinical context. Standard definitions, such as the WHO hemoglobin (Hb) cut-offs stratified by age, sex, and pregnancy status, are valuable for cross-population comparisons, surveillance, and program reporting. However, in HbSS—the commonest severe SCD genotype—the concept of anemia is complicated by a chronically lower "steady-state" Hb driven by ongoing hemolysis [10]. Applying generic cut-offs without acknowledging this baseline shift can inflate apparent anemia prevalence and blur the distinction between chronic anemia that is expected in SCD and anemia that signals acute deterioration. In Ugandan hospital settings, therefore, clinically meaningful measurement should go beyond a single Hb value to include operational indicators that guide urgent decisions. These include severity bands (e.g., Hb <7 g/dL and especially Hb <6 g/dL), and—critically—acute Hb drops from an established steady-state baseline, which may indicate aplastic crisis, sequestration, infection, or bleeding [11]. Symptom-based assessment (pallor, tachycardia, and features of heart failure) adds physiologic relevance, while transfusion metrics (proportion transfused and documented indications) provide a pragmatic endpoint reflecting clinician judgment and resource use. Notably, Northern Uganda inpatient practice explicitly cites transfusion guided by "severe anaemia (Hb <6 g/dL)," underscoring the need to report anaemia prevalence with explicit thresholds and context [12].

Uganda facility-based evidence on anemia burden in SCD patients

Ugandan facility-based data underscore a substantial burden of clinically important anemia among patients with sickle cell disease (SCD), spanning both routine ambulatory follow-up and acute inpatient care. At the Mulago Hospital Sickle Cell Clinic in Kampala, a cross-sectional assessment of 216 children aged 1–18 years (Jan–Feb 2010) deliberately excluded the most profoundly anemic patients (Hb <5 g/dL), yet still revealed marked anemia severity in "stable" clinic attendees. Approximately one-quarter of children had Hb <7 g/dL at assessment (about 25–28%, reported as 20/80 vs 38/136 across HbF strata), meeting a widely used clinical threshold for severe anemia [13]. This pattern indicates that moderate-to-severe anemia is not confined to crises or admissions; rather, it is a common outpatient reality, and the true prevalence is plausibly higher if those excluded with Hb <5 g/dL were counted. Complementing this, a retrospective review of pediatric SCA admissions at Gulu Regional Referral Hospital in Northern Uganda (Jan 2019–Jun 2024) highlights anemia as a dominant driver of inpatient morbidity and resource use: pallor was documented in 19.8% at presentation, while 50.8% required blood transfusion during admission, consistent with practice aligned to severe anemia (Hb <6 g/dL) and acute complications. Taken together, these studies suggest that, beyond the near-universal presence of anemia in HbSS, Ugandan health facilities frequently manage more severe, clinically consequential anemia, particularly during hospitalizations where transfusion needs are common [14].

Drivers of anemia severity in Ugandan SCD patients

In Ugandan patients with sickle cell disease (SCD), anemia severity is driven by more than "baseline" hemolysis from chronic sickling. In malaria-endemic settings, *Plasmodium falciparum* can markedly deepen anemia through a dual hit: accelerated red cell destruction plus inflammatory marrow suppression; clinically, episodes of profound anemia may also be the point at which previously unrecognized SCD is first investigated and diagnosed. Superimposed bacterial infections including pneumonia, invasive bacteremia, and sepsis can further intensify hemolysis while simultaneously blunting erythropoiesis via cytokine-mediated suppression and poor nutritional intake during illness [15]. Episodic aplastic crisis, classically due to parvovirus B19, is especially important because it causes abrupt hemoglobin falls with inappropriately low reticulocyte responses; in many low-resource facilities, limited access to virologic tests and reticulocyte counting risks under-detection and delayed supportive care. Nutritional factors also shape severity: folate requirements rise with high red cell turnover, while iron deficiency may coexist due to dietary limitation or parasitism, yet interpretation is complicated in frequently transfused patients where iron overload and inflammation can mask true deficiency. Helminth infections and chronic inflammatory states contribute to iron-restricted erythropoiesis and impaired marrow efficiency. Over time, progressive renal involvement may reduce erythropoietin production, adding an age-related component to worsening anemia [16].

Clinical implications for Ugandan hospitals

Ugandan hospitals can strengthen SCD care by tightening how anemia is measured, investigated, and treated across clinics and wards. First, standardizing anemia reporting—mean/median hemoglobin (Hb) at admission/discharge, proportions with Hb <7 g/dL and <6 g/dL, reticulocyte status (low vs appropriate), and transfusion rates with clear indications (e.g., severe anemia vs ACS/stroke preparation) will improve comparability between facilities and over time, and allow services to audit outcomes [17]. Second, when patients present with "worse-than-usual" anemia, a pragmatic, resource-adapted minimum work-up should be routine: CBC with indices plus reticulocyte count, malaria testing (RDT/microscopy) when symptomatic or after endemic exposure, and basic hemolysis markers where feasible (bilirubin, LDH). Iron studies should be targeted rather than reflexive, especially where transfusion exposure

is common to avoid inappropriate iron therapy in heavily transfused patients. Third, transfusion stewardship is essential, particularly in regions reporting high inpatient transfusion rates (e.g., Northern Uganda): aligning Hb triggers with locally feasible guidance and consistently documenting indications can reduce inappropriate use and preserve scarce blood for those at highest risk. Finally, expanding access to hydroxyurea could lower anemia burden and complications, but practical barriers in regional hospitals must be addressed [18].

Research gaps and a Uganda-focused roadmap

Uganda's anemia burden, especially severe Hb phenotypes that drive admissions and transfusion demand, still lacks a coherent national evidence base, creating blind spots for planning and evaluation. A priority gap is the absence of harmonized, multi-center prevalence studies that quantify Hb severity bands across Mulago/Kiruddu, regional referral hospitals (Mbale, Mbarara, Gulu, Lira, Arua), and district facilities, enabling benchmarking and catchment-level comparisons. Closely linked is limited understanding of seasonality: rigorous time-series work is needed to map rainy-season malaria peaks against anemia admissions, transfusion utilization, and stock-outs to improve forecasting [19]. Mechanistic attribution is also underdeveloped; combining reticulocyte indices with malaria testing and inflammation/iron markers would better separate hemolysis-dominant anemia from marrow-suppressed states, guiding targeted therapies and stewardship of blood products. Post-transfusion outcomes remain poorly characterized in routine care, including alloimmunization risk, proxies of iron overload, and readmission trajectories like metrics essential for safety and cost-effectiveness. Finally, as hydroxyurea access expands, implementation studies should track shifts in Hb distributions and longitudinal transfusion frequency, identifying real-world responders, adherence barriers, and facility-level determinants of impact. Together, these steps form a pragmatic Uganda-focused roadmap for surveillance, causality, and outcomes-driven policy [20].

CONCLUSION

Uganda's anemia burden, driven in part by severe Hb phenotypes that sustain admissions and transfusion demand, remains underestimated because evidence is fragmented and non-comparable across facilities. This review highlights a practical agenda to close the largest blind spots: harmonized multicenter prevalence work that reports Hb severity bands across national referral, regional, and district hospitals; robust time-series analyses linking rainfall, malaria peaks, anemia admissions, transfusion use, and stock-outs for better forecasting; and improved causal attribution using reticulocyte indices alongside malaria diagnostics and inflammation/iron biomarkers to distinguish hemolysis from marrow suppression. Patient-centered outcomes must also be measured, including post-transfusion reactions, alloimmunization, proxies of iron overload, and readmission trajectories to strengthen safety and value. As hydroxyurea availability expands, implementation studies should track shifts in Hb distributions and longitudinal transfusion frequency, identifying responders, adherence barriers, and facility determinants. Collectively, these steps can translate surveillance into targeted therapy, blood stewardship, and outcomes-driven national policy at scale nationwide.

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