BODY MASS INDEX AND IRON INDICES OF ADULT SICKLE CELL ANEMIA SUBJECTS VISITING LAGOS UNIVERSITY TEACHING HOSPITAL, IDI-ARABA, LAGOS

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BEING A THESIS IN THE DEPARTMENT OF MEDICAL LABORATORY SCIENCE SUBMITTED TO THE COLLEGE OF POST-GRADUATE STUDIES IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE AWARD OF MASTER OF SCIENCE (M. Sc) IN MEDICAL LABORATORY SCIENCE (HAEMATOLOGY AND BLOOD TRANSFUSION SCIENCE) IN UNIVERSITY OF BENIN, BENIN CITY,
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DEDICATION

This work is dedicated to Almighty God and to the Memory of my Late Father and Mother.

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ABSTRACT

Sickle cell anemia (SCA) is a major health problem in Nigeria, contributing greatly to illness and death. Nutrition and iron balance play key roles in how the disease progresses. Body mass index (BMI) affects the general health of SCA patients, while changes in iron levels, whether too low or too high, can worsen anemia and related complications. This study examined the relationship between body mass index (BMI) and iron indices among adult sickle cell anemia (SCA) patients attending the Lagos University Teaching Hospital (LUTH), Idi-Araba, Lagos and to determine their association with disease severity. A cross-sectional analytical design was adopted, involving 45 confirmed HbSS patients and 45 HbAA age- and sex-matched controls. Data on sociodemographic characteristics, pain frequency, and disease severity were obtained using structured questionnaires, the Adult Sickle Cell Quality of Life Measurement Information System (ASCQ-ME), and the Modified Disease Severity Scoring System for SCA. Anthropometric variables were measured following standard protocols, and BMI was classified based on WHO criteria. Blood samples were analyzed for full blood count, serum iron, serum ferritin, and total iron-binding capacity (TIBC) using standard ELISA and spectrophotometric techniques. Disease severity score was obtained for each Hbss subject by summing up the scores for crisis rate, number of complications and degree of anemia. HbSS total scores of <3 were considered to have mild anemia. Those with score of > 3 but < and =7 were taken have moderately anemia while subjects with scores > 7 were deemed to have severe anemia. Most participants were young adults. In the SCA group, 46.7% were aged 18–25, while 40% of the control group fell within this range. Participants aged 26–35 constituted 20% of the SCA group and 17.8% of controls. Those aged 36–45 represented 22.2% of the SCA group and 28.9% of the controls. Smaller proportions were observed in the 46–55 age bracket, with both groups recording 8.9%. Only a few participants were 56 years and above, comprising 2.2% of the SCA group and 4.4% of the control group. The SCA patients had significantly lower hemoglobin (HGB), packed cell volume (PCV), and red blood cell (RBC) counts compared to controls ($p < 0.001$). Mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) were significantly higher in SCA subjects ($p < 0.001$). Ferritin concentrations were markedly elevated ($p < 0.001$), whereas TIBC was significantly reduced ($p < 0.001$). Serum iron levels did not differ significantly between groups ($p > 0.05$). BMI distribution indicated that 28 (62.2%) of the SCA subjects had normal weight, 13 (28.9%) were underweight, and 4 (8.9%) overweight. Lower BMI correlated with more severe anemia and higher disease severity scores ($p < 0.05$). Painful crises were common, with vaso-occlusive crisis and acute chest syndrome observed in all patients, while mild to moderate anemia predominated (75.6%). In conclusion, this study demonstrates that low TIBC and elevated serum ferritin are frequent among adult SCA patients, reflecting chronic inflammation and increased iron stores within the reticuloendothelial system. Nutritional monitoring and iron indices evaluation should be integral to routine care for SCA patients to reduce disease complications and improve quality active life.

CHAPTER ONE

INTRODUCTION

1.1 Background to the Study

Sickle cell anemia is the most common inherited blood disorder (Onoja *et al*., 2020). It is caused by a single mutation in the β-globin gene, which leads to the production of an abnormal hemoglobin known as hemoglobin S (HbS). Under conditions of reduced oxygen, such as stress, infection, dehydration, and lower temperatures, HbS polymerizes, causing hemoglobin molecules to stick together. This result in the formation of sickle-shaped red blood cells, leading to chronic hemolytic anemia that requires blood transfusions, pain crises, and can cause damage to various organs (Mangla *et al*., 2024). Individuals with one sickle cell mutation have sickle cell trait, while those with mutations in both hemoglobin genes have sickle cell anemia (HbSS).

According to a global study conducted from 2000 to 2021, about 8 million people were living with sickle cell disease, and more than half a million babies were born with the condition (GBD, 2023). This represents a rise of 13.7%, with over 75% of sickle cell disease births occurring in sub-Saharan Africa. Almost half (44%) of the global incidence of sickle cell disease at birth was accounted for by six countries: Equatorial Guinea, the Republic of Benin, Burkina Faso, Nigeria, Sierra Leone, and Togo. Evidence showed that Nigeria has the largest population of individuals affected by sickle cell disease (Adigwe *et al*., 2023).

In Nigeria, out of an estimated 250,000 babies are born with sickle cell disease annually, 70-90% die before age 5, only few reach adolescent (Stephen *et al*., 2018).

Varying prevalence has been recorded across Nigeria: with prevalence ranging from 1.63% to 5% in the south-east (Diwe *et al*., 2016; Ogbonna *et al*., 2022), 2.14% in South-south (Kingsley *et al.,* 2019), 2.4% in South-west (Taiwo *et al*., 2011) and 2.69% in Northern Nigeria. (Inusa *et al*., 2015).

Several studies have shown that these relative differences in sickle cell disease prevalence could be attributable to the disparities in awareness, knowledge, perceptions and cultural practices in respect to pre-genetic counseling in the multi-ethnic groups that make up Nigeria (Uche *et al*., 2017; Ogamba *et al*., 2020, Adigwe, 2022). This study however focuses specifically on adults with sickle cell anemia (SCA; HbSS genotype)**.**

Under deoxygenated state, sickle-shaped red blood cells occlude capillaries and prevent tissue oxygen delivery, leading to acute and chronic pain, severe anemia, kidney dysfunction, acute chest syndrome, stroke, and other cardiovascular diseases (Rees *et al*., 2010; Jain *et al*., 2019). The low levels of oxygen in blood and tissue hypo-fusion lead to tissue impairments which can affect almost all systems of the body and is associated with retarded growth, poor development, and poor nutritional status that results in deterioration in different anthropometric variables such as body mass index, skeletal development, and late puberty (Odetunde *et al*., 2016). These clinical manifestations also appear to be associated with changes in physical capacity, higher basal metabolic rate, reduced levels of hemoglobin, vaso-occlusive crisis, pulmonary vascular disease, and myopathy that can lead to sedentary behavior (Van Beers *et al*., 2014).

Body Mass Index (BMI) is calculated as weight in kilograms divided by height squared in meters. BMI is categorized into underweight, normal, overweight, and obese. It is a pointer to health status. It is directly associated with various cardiovascular risk factors (Nuttall, 2015). The World Health Organization (WHO, 2021) classifies BMI into several categories: underweight (BMI < 18.5), normal weight (BMI 18.5-24.9), overweight (BMI 25-29.9), and obese (BMI ≥ 30). As body mass index increases, so do blood pressure, low-density lipoprotein, cholesterol, sleep apnea, liver disease as well as different types of cancers (Shmerling, 2023). Obesity is also associated with reduced quality of life, mental health issues, and increased mortality. The higher the body mass index the greater the risk of these complications (WHO, 2021). An increasing prevalence of overweight and obesity had been reported among patients with SCA. (Eke *et al.,* 2015). Adolescents with sickle cell anemia are likely to benefit from interventions to decrease obesity risk factors so as not to worsen the symptoms and complications of the disease.

Low body mass index has been reported among SCA patients and is linked to adverse outcomes (Odetunde *et al.,* 2016). Odetunde *et al.,* reported a 48% prevalence of underweight among patients with SCA compared to 13% in children with Hb AA. Malnutrition in SCA can result from increased metabolic demands, chronic inflammation, and poor dietary intake. Research indicates that underweight SCA patients often experience more severe and frequent vaso-occlusive crises, growth retardation, osteoporosis and delayed puberty (Van Beers *et al*., 2014). Malnutrition also weakens the immune system, making patients more susceptible to infections, which can aggravate the disease. A study by Barden *et al.*, (2002) demonstrated that

children with SCA and lower BMI had reduced fat stores and lean body mass, which correlated with higher rates of hospitalization and complications. Additionally, poor nutritional status can impair wound healing and recovery from illness, further contributing to a poor prognosis (Enyuma *et al*., 2019).

Adequate nutrition supports the body's increased metabolic needs and helps manage chronic inflammation and oxidative stress associated with SCA. Proper nutritional status is essential for maintaining immune function, reducing the frequency of vaso-occlusive crises, and preventing growth delays in pediatric patients.

Although, not usually common, overweight and obesity can also negatively impact SCA management. Obesity increases the risk of comorbid conditions such as hypertension, diabetes, and obstructive sleep apnea, which can further complicate SCA by adding cardiovascular strain and enhancing systemic inflammation (Hall *et al*., 2010; Heymsfield and Wadden, 2017). Excess adiposity can also increase the frequency and severity of vaso-occlusive crises and contribute to organ damage, particularly in the kidneys and liver. Tambe (2017) found that overweight and obese Maintaining a normal BMI is associated with better clinical outcomes in SCA. Patients with a normal BMI generally have fewer complications and a better overall quality of life (WHO, 2020). children with SCA had reduced pulmonary function compared to those with normal BMI, suggesting that excess weight can impair respiratory health, which is critical in SCA patients. Another study found out that group with BMI $\geq$ 30 kg/m^2 had a 20.8% increased risk of SCA (Yun *et al.,* 2023).

Furthermore, obesity-related inflammation can exacerbate the chronic inflammatory state seen in SCA, worsening the overall prognosis.

Elevated BMI is strongly associated with an increased risk of thrombosis. Obesity leads to hypercoagulable state and increased secretion of plasminogen activator inhibitor (PAI)-1 and thrombin activatable fibrinolysis inhibitor (TAFI) which increases the likelihood of blood clot formation (Vilahur *et al.,* 2017). This is due to several factors, including increased levels of pro-coagulant factors, decreased fibrinolytic activity, low grade inflammation, increased oxidative stress and endothelial dysfunction as well as disturbance of lipids and glucose tolerance. Adipose tissue, particularly visceral fat, secretes inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), which can promote thrombogenesis (Blokhin and Lentz, 2013). Hyperlipidemia, elevated levels of triglycerides, low-density lipoprotein (LDL) and cholesterol in obese individuals contribute to atherogenesis, which can further increase the risk of arterial thrombosis in sickle cell anemia patients. It thus implies that obesity may further increase the risk of thrombosis in patients with sickle cell disease.

The relationship between Body Mass Index (BMI) and iron indices in adult patients with Sickle Cell Anemia (SCA) is complex, influencing the degree of anemia and overall health outcomes. SCA patients often face unique challenges regarding iron metabolism due to their disease pathology and treatment regimens, such as frequent blood transfusions which poses the risk of iron overload.

There is growing evidence that iron metabolism is altered in obese people. It has been reported to leads to systemic iron deficiency and tissue iron overload (Zhao *et al*., 2015; Qiu *et al*., 2022). Adipose tissue in obese people express increased levels hepcidin and haemojuvelin, proteins which is associated with iron deficiency (Qiu *et al*., 2022).

Iron indices influences the risk of thrombosis. Both iron deficiency and iron overload have been implicated in thrombotic events, though the mechanisms may differ. Excessive iron can catalyze the formation of reactive oxygen species (ROS), leading to oxidative stress and endothelial injury which promotes thrombosis (Wun and Brunson, 2016), while, severe iron deficiency can also alter platelet function and increase the risk of thrombosis. Iron deficiency is associated with increased platelet count and altered platelet reactivity, which can enhance clot formation (Savarese *et al*., 2023).

In sickle cell anemia, iron indices is often disrupted. SCA patients frequently experience episodes of hemolysis, releasing free hemoglobin and iron into the circulation. Excessive iron mainly affects the heart, lungs and endocrine glands. Hepatic cirrhosis from excessive iron is a major cause of death in patients with sickle cell anemia (Remacha *et al*., 2013). This environment is favorable for thrombus formation (Rees *et al*., 2010).

Obesity can exacerbate the complications of SCA. Elevated BMI in SCA patients is associated with increased morbidity due to higher rates of vaso-occlusive crises, which are linked to thrombosis. Obesity further complicates the management of SCA

by increasing the risk of conditions such as hypertension and diabetes, which can exacerbate vascular complications.

SCA patients often exhibit altered iron metabolism due to chronic hemolysis and frequent blood transfusions. These factors can lead to iron overload, which, as mentioned earlier, can increase oxidative stress and thrombotic risk. Conversely, iron deficiency can occur due to poor nutritional status or increased demand, potentially affecting thrombotic risk through altered platelet function.

The interplay between BMI, iron indices, and thrombosis in SCA patients is complex. Elevated BMI can increase inflammation and hypercoagulability, while disrupted iron metabolism can further contribute to oxidative stress and endothelial damage, amplifying the effect of clot. Understanding these relationships is crucial for managing SCA patients, as both nutritional and weight management, along with careful monitoring of iron levels, will be essential to mitigate thrombotic risk.

1.2 Statement of the Problem

In Nigeria, out of an estimated 250,000 babies are born with sickle cell disease annually, 70-90% die before age 5, only few reach adolescent (Stephen *et al*., 2018). The prevalence ranges from 1%-5% (Thomas *et al.,* 2013; Nwabuko and Oko, 2015). A prevalence of 2.4% was reported in South Western Nigeria by (Taiwo *et al*., 2011) for HbSS genotype. Another study, reported that 42.9% of adult SCA had a BMI of >25kg/m^2 (Ibemere *et al*., 2022).

Enhancements in medical care and quality of life have led to increased longevity and hence at risk with other co-morbidity like obesity.

BMI is negatively correlated with iron indices, meaning that a higher BMI is associated with a reduced iron load and an increased risk of iron deficiency anemia. Consequently, patients with sickle cell anemia (SCA) with increased BMI are at a higher risk of worsening anemia with antecedent hypoxia and more severe disease outcomes.

1.3 Justification of the Study

SCA affects 1-5% of people in Nigeria. Overweight and obesity is also seen in 25.6% and 14.5% respectively in Nigerian adults sickle cell anemia and has been reported among adults with SCA (Chukwuonye *et al.,* 2022).

Understanding the correlation between BMI, iron indices, and thrombosis is critical, especially in patients with complex conditions like sickle cell anemia. Elevated BMI and iron dysregulation both contribute to an increased risk of thrombotic events, underscoring the need for comprehensive management strategies that address weight, nutritional status, and iron levels to improve patient outcomes.

This will help to develop a protocol for early identification to increasing BMI and early recognition of iron deficiency among these patients. With ongoing care, patients can go on to live a full active life.

The findings will be valuable for healthcare providers in tailoring interventions to improve the health outcomes of SCA patients based on their BMI and iron indices as well as policy makers by providing support in ensuring that the test is done routinely for sickle cell anemia patients.

1.4 Aim of the Study

The aim of this study is to determine the body mass index and iron indices of adult sickle cell anemia subjects visiting Lagos University Teaching Hospital, Idi-Araba, Lagos.

1.4.1 Specific Objectives

The specific objectives of this study are:

a. to determine the levels of serum iron, serum ferritin and total iron binding capacity (TIBC) in the study population.
b. to determine the mean body mass index among adult SCA patients in LUTH, Idi-Araba.
c. to carry out FBC analysis in all the samples.
d. to determine the relationship if any between BMI and iron indices in patients with SCA.
e. to correlate the body mass index and the levels of iron indices to the severity of sickle cell anemia among the study population.

1.5 Research Questions

The following research questions were used for this study;

a. What is the iron indices of SCA patients?
b. What is the pattern of BMI in patients with SCA?
c. Is there any different between the full blood count (red blood cell, White blood cell and Platelet) of sickle cell anemia subjects and control groups?

d. Is there any association in the body mass index and iron indices among sickle cell anemia patients visiting LUTH, Idi-Araba?
e. What is the impact of BMI and iron indices on SCA?

1.6 Research Hypothesis

The following Hypothesis were used for this study;

Ho; There is no association between body mass index and iron indices in patients with SCA.

Ha; There is association between body mass index and iron indices in patients with SCA.

Ho; There is no association in body mass index and iron indices and severity of disease among the sickle cell anemia patients in LUTH, Idi-Araba.

Ha; There is association in body mass index and iron indices and severity of disease among the sickle cell anemia patients in LUTH, Idi-Araba.

CHAPTER TWO

LITERATURE REVIEW

2.1 Sickle Cell Anemia

According to the Oxford dictionary, sickle cell anemia, a hemoglobin disorder, is a severe hereditary form of anemia in which mutated hemoglobin genes are inherited from both parents. Sickle cell anemia is a specific form of sickle cell disease in which there are two forms of 'S' sickle cell gene. Normal Hemoglobin (HbA): composed of two α and two β chains ($\alpha_2\beta_2$), while the Mutated Hemoglobin (HbS): is composed of two α and two mutated β chains ($\alpha_2\beta$^S).

Sickle cell anemia is characterized by changes in the shape of the red blood cell from a smooth doughnut to a crescent shape. The misshapen cells lack plasticity and cause blockage to small vessels, impairing blood flow. This condition leads to shortened red blood cell survival and subsequent anemia. Poor blood oxygen levels and blockage of blood vessels in people with sickle cell anemia lead to chronic acute pain syndromes, severe bacterial infections, and tissue death. The life span of sickle cell blood lasts only 10-20days, unlike normal blood cells that have a duration of 90 -120 days (Mangla *et al.,* 2024). The body produces new red blood cells to replace the old cells. However, in sickle cell anemia, the body finds it difficult to keep up with the rate at which cells are being destroyed, leading to anemia that causes tiredness and less energy (Jain *et al.,* 2019).

Other types of sickle cell disease include;

I. Hemoglobin SC (Hb SC)

Hb SC develops in people who inherited one ‘S’ from one parent and an irregular ‘C’ type gene from the other parent. This is milder than Hb SS.

II. Hemoglobin S beta thalassemia (Hb S)

This condition occurs when an ‘S’ gene from one parent and a gene for another type of anemia called beta thalassemia from the other parent are inherited

III. Hb SD, Hb SE, or Hb SO

These are uncommon forms of sickle cell disease where an ‘S’ gene from one parent and a ‘D’,’E’ or ‘O’ hemoglobin gene from the other parent. The severity varies here.

IV. Hemoglobin AS (Hb AS)

Also known as sickle cell trait. In this case, a person inherits the ‘S’ gene from one parent and a normal gene from the other. They show no symptoms but are carriers of the sickle cell gene and can pass it to their children.

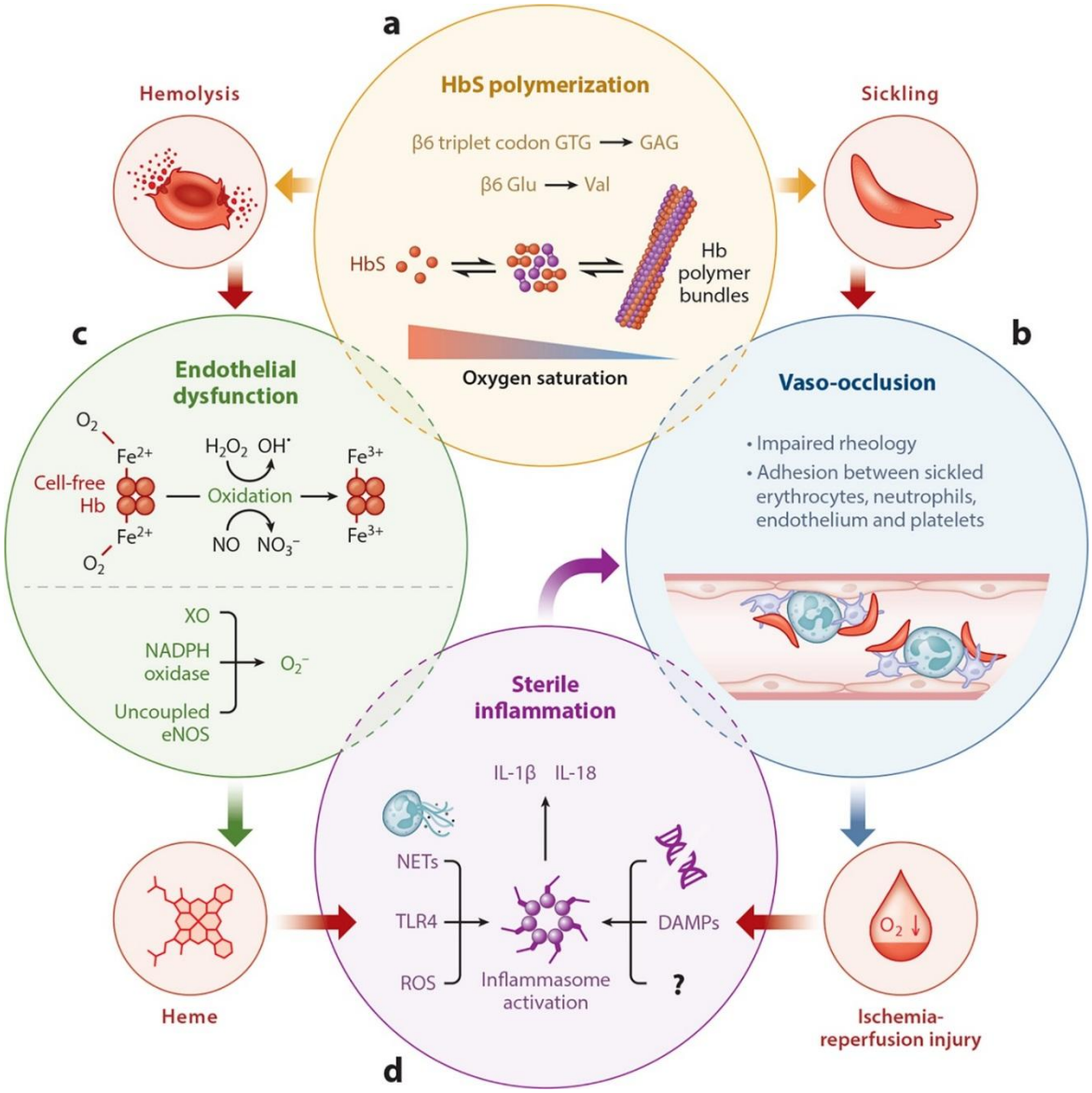

Figure 2.1 Molecular Pathophysiology of Sickle Cell Anemia (Sundd *et al.,* 2018).

2.2 Molecular Pathophysiology of Sickle Cell Anemia

A single-nucleotide mutation in the β-globin gene causes the replacement of glutamic acid with valine at the sixth position of the β-globin chain (De Franceschi *et al.,* 2011). Upon low oxygen, the mutated hemoglobin (HbS) molecules aggregate into polymerized structures. These polymer chains cause the red blood cells to sickle (clockwise). The polymerization is reversible initially, but repeated cycles lead to membrane damage.

Key Steps in the Pathophysiology

2.2.1 Red Blood Cell Sickling

Sickled cells have the following properties:

- Rigid and inflexible: This implies they can't pass easily through the microvasculature.
- Fragile: This means they are prone to hemolysis, leading to chronic hemolytic anemia.
- Shortened lifespan: a lifespan of approximately 10–20 days compared to 90 -120 days for a normal erythrocyte.

These properties lead to altered blood flow dynamics and clumping of sickled cells with neutrophils, platelets, and endothelial cells, resulting in blood flow obstruction, known as vaso-occlusion. Vaso-occlusion (crises) leads to ischemia-reperfusion (I-R) injury, pain, and organ damage (brain, lungs, kidneys) complications (Gladwin *et al.,* 2014; Novelli *et al.,* 2014; Saraf *et al.,* 2014). Anemia and intravascular hemolysis

lead to pulmonary vascular disease and diastolic heart dysfunction, contributory factors to morbidity and death (Gladwin *et al.,* 2016).

The intrinsic rate of hemolytic anemia is relatively stable within an individual patient with SCA under steady-state (non-crisis) conditions and is determined mainly by the hemoglobin genotype (Hb S, C, etc.) and Hb F levels (Milton *et al.,* 2013; Nouraie *et al.,* 2013).

2.2.2 Endothelial Activation and Inflammation

- Chronic hemolysis releases free hemoglobin and reactive oxygen species, which lead to endothelial dysfunction.
- Damaged endothelium expresses adhesion molecules (e.g., VCAM-1, ICAM-1, E- E-selectin), promoting interaction with sickled erythrocytes, leucocytes, and platelets.

Due to the releases of free hemoglobin into the bloodstream, oxygenated hemoglobin (Fe2+) impairs endothelial function by depleting nitric oxide (NO) reserves in the endothelial cells, converting them to nitrate (NO3−) and methemoglobin (Fe3+). Alternatively, hemoglobin can interact with H^2O^2 in the Fenton reaction to generate hydroxyl radicals (OH•) and methemoglobin (Fe3+). Additionally, enzymes such as NADPH oxidase, xanthine oxidase (XO), and uncoupled endothelial nitric oxide synthase (eNOS) produce reactive oxygen species (ROS), further impairing endothelial function. Methemoglobin (Fe3+) breaks down to release free heme (counterclockwise), which is a significant damage-associated molecular pattern (DAMP) linked with red blood cell destruction.

The generation of reactive oxygen species (ROS), activation of Toll-like receptor 4 (TLR4), formation of neutrophil extracellular traps (NETs), release of tissue or cell-derived DAMPs, DNA, and other unknown factors triggered by free heme or I-R injury can contribute to sterile inflammation. This inflammation is mediated through the inflammasome pathway, activating vascular and inflammatory cells to release IL-1β and IL-18. Lastly, sterile inflammation exacerbates vaso occlusion through a positive feedback loop, enhancing the adhesion of neutrophils, platelets, and endothelial cells.

As patients with SCA live longer in high-income countries, the chronic impact of sustained hemolytic anemia and episodic vaso-occlusive events results in the progressive development of end-organ complications (Bartolucci *et al.,* 2012; Gladwin *et al.,* 2014; Saraf *et al .,* 2014).

2.2.3 Vaso-Occlusion

Vaso-occlusion in sickle cell anemia results from a complex interaction of sickled red cells, endothelial activation, white blood cells, platelet adhesion, and inflammatory processes. This cascade leads to intermittent and chronic microvascular obstruction, which is central to the disease's morbidity. The accumulation of cells leads to blockage of blood flow in small vessels.

This reduces oxygen delivery to tissues (ischemia), perpetuating more sickling and a vicious cycle of hypoxia and further occlusion. Tissue ischemia and reperfusion injury- lack of oxygen causes pain, cell death, and organ damage. When blood flow returns (reperfusion), it causes oxidative stress and inflammation, worsening tissue

injury. This condition often necessitates urgent medical intervention for affected individuals (Manwani *et al.,* 2013). Research using transgenic humanized

SCD mouse models and in vitro flow chamber studies has provided key insights. Vaso-occlusion in sickle cell anemia involves several factors. These include altered blood flow, increased adhesion of sickled red blood cells to inflammatory cells and the endothelium, and activation of the coagulation system (Zhang *et al.,* 2016). Blood flow characteristics, or rheology, are influenced by hematocrit levels, plasma viscosity, and the flexibility of red blood cells (Barabino *et al.,* 2010). Elevated plasma viscosity, which is caused by chronic hemolysis and the reduced deformability of sickled erythrocytes due to hemoglobin polymerization and cell dehydration, impairs the passage of blood through capillaries and venules in tissues with high oxygen requirements (Barabin *et al.,* 2010).

Due to chronic anemia, the bone marrow experiences stress reticulocytosis, leading to the premature release of immature red blood cells or reticulocytes. Patients with sickle cell anemia typically exhibit elevated baseline levels of neutrophils, monocytes, and platelets.

Increased concentrations of circulating neutrophil-platelet and monocyte-platelet aggregates in the blood of individuals with SCA have been shown to correlate with disease severity (Curtis *et al.,* 2015; Wongtong *et al.,* 2015).

Furthermore, thrombocytopenia is associated with the progression from vaso-occlusive crisis (VOC) to acute chest syndrome (ACS), a potentially fatal pulmonary complication in SCA patients (Chaturvedi *et al.,* 2016). This association suggests that

platelet sequestration at sites of vaso-occlusion plays a critical role in the development of ACS (Alhandalous *et al.,* 2015).

Evidence shows that vaso-occlusive crises (VOC) are frequently triggered by inflammatory or environmental stimuli, including cold temperatures, dehydration, excessive exercise, tobacco smoke, infection, hypoxia, high altitude, airplane travel, and other unidentified factors (Novelli and Gladwin, 2016). The intrinsic rate of hemolytic anemia remains relatively stable in individuals with sickle cell anemia during steady-state (non-crisis) conditions (Nouraie *et al.,* 2013). Patients with high rates of hemolysis generally have lower steady-state hemoglobin levels and are more likely to develop vascular injury and progressive organ dysfunction with age.

These complications manifest as pulmonary hypertension, diastolic left heart dysfunction, and renal impairment, including proteinuria, albuminuria, and chronic kidney disease (Gladwin *et al.,* 2014; Saraf *et al.,* 2014).

2.3 Signs and Symptoms of Sickle Cell Anemia

The majority of newborns with sickle cell anemia do not have symptoms until about six (6) months old. Symptoms vary from person to person and can change over time. Symptoms depend on how sickle cell anemia affects the individual's health. Some show symptoms occasionally, while others show symptoms very often. Sickled cells break apart easily and die, leading to a shortage of healthy red blood cells. Early symptoms of SCA include jaundice (yellowing of the skin and whitening of the eyes), extreme tiredness, dactylitis, and swelling of the hands and feet. Sickle cell anemia experience pain in different ways. The pain can be in the form of acute or severe

paincrisis, pain from organ damage, joint pain, chronic pain, or priapism (prolonged painful erection). Periodic episodes of extreme pain are the major symptoms of sickle cell anemia. Other symptoms include frequent infections, delayed growth or puberty, and vision problems. Also, life-threatening anemia can be caused by splenic sequestration crisis, fever of more than 38.5^{o} C needs attention from healthcare providers, as well as acute chest syndrome (ASH, 2023).

However, chronic anemia results in bone marrow compensation being inadequate to match the rate of destruction (reticulocytosis); baseline hemoglobin levels (6-10g/dl); nutrient deficiencies (folate) due to increased erythropoiesis; stroke; avascular necrosis especially of the femoral head; chronic organ damage such as brain, kidneys and spleen (Connes *et al.,* 2014; Nouraire *et al.,* 2013).

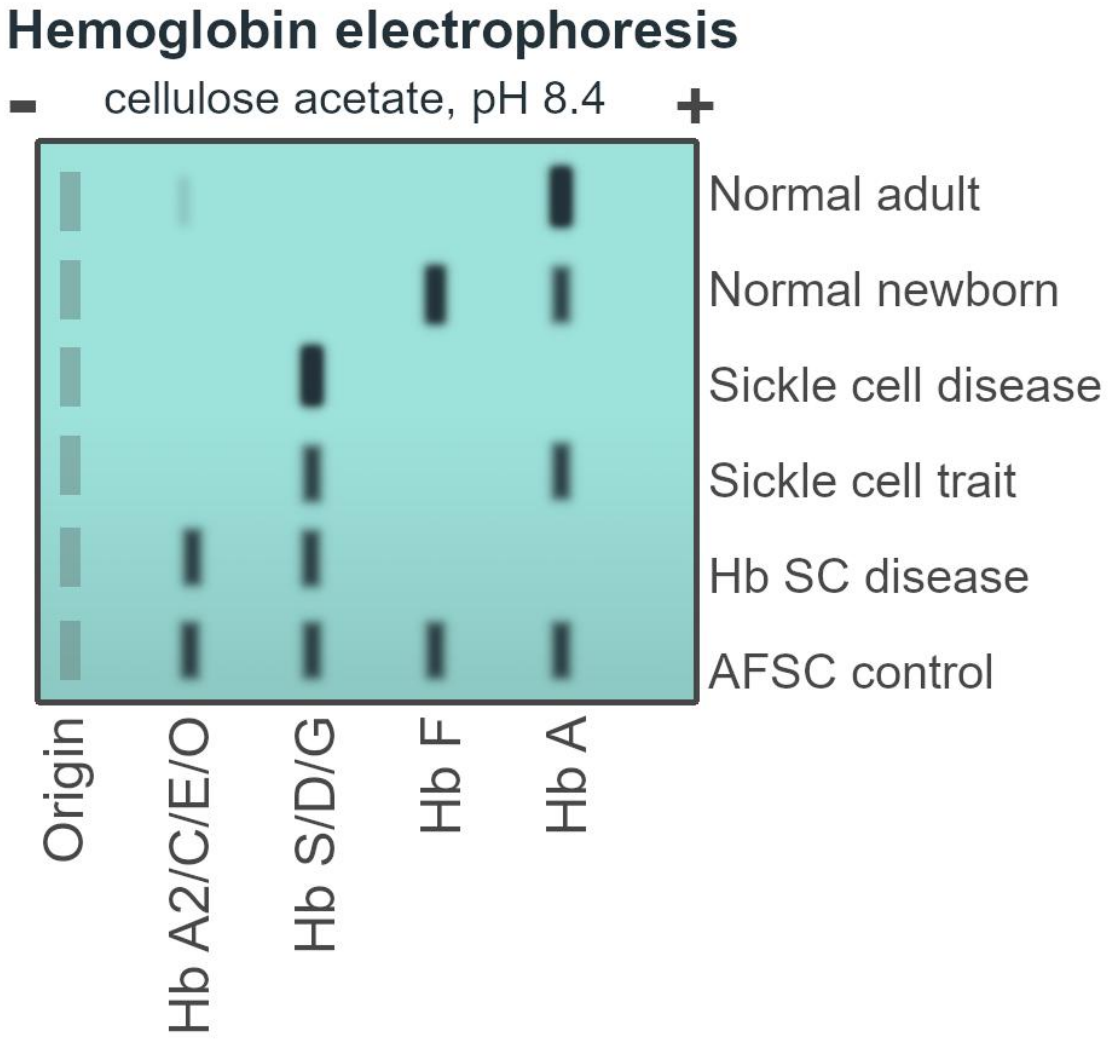

Figure 2.2 Hemoglobin Electrophoresis (Keohane *et al.,* 2015; McPherson and Pincus 2017).

2.4 Diagnosis of Sickle Cell Anemia

Sickle cell anemia is diagnosed by using hemoglobin electrophoresis or high-performance liquid chromatography. The test identifies and measures different types of hemoglobin in red blood cells, including the abnormal hemoglobin that causes sickle cell anemia.

A blood test is used to determine HbS protein and its quantification. Samples are introduced at the origin point and the various types of hemoglobin migrate from an area of negative (-) to positive (+) charge. The speed of migration is based on the molecule's charge. The migration patterns of hemoglobin variants is from slowest to fastest: Hemoglobin A2/C/E/O Hemoglobin S/D/G Hemoglobin F Hemoglobin A (Keohane *et al.,* 2015); McPherson and Pincus 2017).

Gene testing can also be used to determine when one or two copies of HbS are present. Other blood tests may also be done. Newborns are screened for sickle cell disease as part of a group of screening tests. The majority of the children start showing symptoms around 5 months. Also, family history, the child's medical history, and physical examination can be used in the diagnosis.

Diagnostic Markers of Hemolysis in Sickle Cell Anemia
Marker Expected Finding
Hemoglobin ↓ Low (6–10 g/dL)
Reticulocyte count ↑ Elevated
LDH ↑ Elevated
Haptoglobin ↓ Undetectable
Bilirubin (indirect) ↑ Elevated
Peripheral smear: Sickled cells, target cells, Howell-Jolly bodies

(Kato *et al.,* 2018)

2.5 Prevalence of Sickle Cell Disease (SCD)

Globally, approximately 5% of the world's population carries trait genes for hemoglobin disorders (sickle cell disease and thalassemia) (WHO, 2024). Sickle cell disease occur in all races and ethnicities. Sickle cell anemia is more common in tropical regions. Predominates in sub-Saharan Africa, Congo, Gabon, Ghana, and Nigeria, with a prevalence of 20-30%. Uganda has 45% (WHO, 2024). People at greater risk are of African descent, Hispanic births from Central and South America, Eastern heritage, Indian heritage, and individuals of Mediterranean descent (CDC, 2024).

Also, it states that sickle cell disease affects approximately 100,000 Americans: occurs in about 1 out of every 365 African American births and 1 out of every 16,300 Hispanic American births.

According to a report from Global Burden Distribution (GBD), total sickle cell disease deaths accounted for 2.2% (1.5-3.0) of all deaths in children younger than 5years and 4.3% (3.5-5.6) of all deaths in individuals aged 15-49years. Reports also showed that the female death rate was higher than the male (3.90 and 3.84 million), respectively, in 2021. However, due to paucity of data related to sickle cell disease, incidence, prevalence, and death rate make it difficult to truly estimate the disease burden, especially as these data are sparse in areas with the highest sickle cell disease prevalence (GBD, 2023). These sickle cell diseases are considered Neglected Tropical Diseases (NTDs), the morbidity and mortality are entirely preventable. Sickle cell

disease imposes a great and often far deadlier burden than its textbook description. It was found that the average life expectancy for publicly insured individuals with sickle cell disease in the U.S.A. was 73.5 years for males, and lower than that of females at 79.3 years (ASH, 2023).

In Nigeria, annually, approximately 250,000 babies are born with SCD, 70-90% die before their fifth birthday, and only a few reach adolescence (Stephen *et al.,* 2018). A review of the geographical spread of sickle cell in Nigeria showed a prevalence of 1.63% to 5%. These were stated in a retrospective cross-sectional study in South-Eastern Nigeria (Ogbonna *et al.,* 2022). Diwe *et al.,* (2016), who conducted another study in Imo State, recorded a prevalence of 5%. A prevalence of 2.4 was reported in South-Western, Nigeria by (Taiwo et al., 2011) for HbSS genotype. In Northern Nigeria, 2.69%, out of 269 local communities based study screened children was recorded as prevalence (Inusa *et al.,* 2015). In Southern South Nigeria, Calabar, the prevalence of 2.14% was observed (Kingsley *et al.,* 2019).

Several studies have shown that these relative differences in sickle cell disease prevalence are attributable to disparities in awareness, knowledge, cultural practices, and perceptions in pre-genetic counselling in the multi-ethnic groups that make up Nigeria (Uche *et al.,* 2017; Ogamba *et al.,* 2020; Adigwe, 2022).

2.6 Body Mass Index and Sickle Cell Anemia

The World Health Organization (WHO, 2021) classified body mass index into different categories: underweight (BMI < 18.5), normal weight (BMI 18.5-24.9), overweight (BMI 25-29.9), and obese (BMI >=30) Kg/m².

The relationship between body mass index (BMI) and sickle cell anemia (SCA) is influenced by Interactions between genetic, metabolic, nutritional, and other socioeconomic factors.

A United States report found that 19%–22% of children with SCD were either overweight or obese (Zivot *et al.,* 2017).

According to 2013–2016 National Health and Nutrition Examination Survey (NHANES) data in adults over 20 years old in the United States, the overall prevalence of overweight and obesity, was 70.9% (Centers for Disease Control and Prevention, 2018; CDC, 2021). But despite the known proportion, the prevalence of overweight or obesity for adults with SCA remains unclear (Zivot *et al.,* 2017; Hall *et al.,* 2018; Ogunsile *et al.,* 2019).

In sub-Saharan Africa where 75% of all children with SCA are born undernutrition, childhood mortality is common, though improved with better care, is still excessive (Kato *et al.,* 2018; Oron *et al.,* 2020). Notably, in the Stroke Prevention in Nigeria (SPRING) trial, the mortality rate among 5–12-year-olds with SCA was far higher than contemporary U.S. figures.

The standard undernutrition indicators (underweight, stunting, wasting) were not assessed, despite Weight-for-age z-score being a sensitive marker of acute and chronic undernutrition and a strong predictor for early childhood mortality. This shows that there is a bidirectional relationship between abnormal BMI (undernutrition or obesity) and disordered iron indices (deficiency or overload). Hence, correlate strongly with SCA severity, morbidity, and mortality (Klein *et al.,* 2023).

Consequently, routine nutritional screening (BMI/weight-for-age) alongside iron assessment (ferritin and transfusion history) should be integral to clinical management and risk stratification in SCA.

Historical data indicating that many SCA patients were underweight (Ibemere *et al.,* 2023). Another study also indicating a prevalence of overweight and obesity (Team and Niblett, 2015).

A study of 100 participants with sickle cell anemia (Hb S and Hb A) found that cases had a lower mean hematocrit (24.86%) compared to controls (38.20%). The mean (SD) body weight was 54.39 kg for cases and 60.48kg in controls. The Body mass index was lower in cases than in controls (19.58 vs. 21.48 kg/m²; Asafa *et al.,* 2022). The mean hematocrit (24.86%) matched earlier findings among similar SCA individuals (Akinbami *et al.,* 2012). Low hematocrit in sickle cell anemia results from ongoing hemolysis and a reduced erythropoietin response that does not align with the severity of anemia (Kato et al., 2018). Low body mass index in SCA has also been observed in previous studies of young Nigerian adults (Dosunmi *et al.,* 2016; Ogunobi *et al.,* 2016). This pattern is linked to chronic anemia, poor nutrient intake, absorption issues, metabolic problems, and reduced appetite during sickle cell crises (Oguanobi *et al.,* 2016).

A retrospective study carried out in Republic of South Korea on the impact of body mass index on the risk of SCA and the influence of risk factors (age, sex, social habit and metabolic disorders) shows that the obese group (BMI $\geq$ 30kg/m^2) had 20.8% increased risk of sickle cell anemia compared with the normal body weight group

(18.5-24.9kg/m^2). Conversely, after adjustments for risk factors, BMI was not associated with SCA risk. Findings suggest that obesity itself is not an independent risk factor for SCA, but a surrogate marker of metabolic disorders and people's demographics (Kim *et al.,* 2023. Although the consequences of SCA events are highly dependent on the geographical accessibility of emergency medical services and the degree of literacy of citizens. The majority of SCA events impose considerable socioeconomic costs on their victims and family members (Myat *et al.,* 2018).

Clarifying whether obesity is independently associated with SCA or is indirectly associated with SCA through the influence of metabolic disorders is important for the prevention of SCA. If coexisting metabolic disorders are the culprit risk factor for SCA, weight reduction itself may not be sufficient to effectively prevent SCA, and concomitant management of metabolic disorders such as hypertension, sleep apnea, and diabetes may be more important (WHO, 2021; Shmerling, 2023).

2.7 BMI and Iron Profile in Sickle Cell Anemia

In addition to the hypermetabolic demands of SCA, iron indices whether deficiency or overload plays an important role in shaping BMI outcomes and influencing disease severity. While some studies show a potential for better health outcomes with normal-to-above-normal BMI in children and a possible association between high BMI and certain complications like hypertension and possibly increased pain in adults, the overall impact on disease severity and the need for routine BMI monitoring and weight management interventions are areas of active research.

Underweight individuals often have diets that lack sufficient iron, leading to reduced iron stores and lower hemoglobin levels. Studies have shown that underweight populations, particularly in low-resource settings, frequently exhibit higher rates of iron deficiency anemia due to inadequate dietary iron intake and poor nutritional status.

Malnutrition associated with low BMI can impair iron absorption and utilization. Essential nutrients required for optimal iron metabolism, such as vitamin C and proteins, might be deficient in underweight individuals, further exacerbating iron deficiency. A study conducted on children and adolescents highlighted that those with lower BMI had significantly lower serum ferritin levels, indicating depleted iron stores (Qui *et al.,* 2022).

Low BMI has been associated with increased frequency of vaso-occlusive crises (VOCs), higher rates of hospitalization, and poorer overall health outcomes, while iron deficiency further exacerbates these risks by contributing to anemia and metabolic complications. Similar associations have been reported in other chronic conditions such as asthma, where abnormal BMI and anemia were linked with greater hospital admissions. In a recent study, most participants were older adults, with a high prevalence of obesity and mild anemia. Those who were obese and those with anemia experienced a higher frequency of asthma-related hospitalizations compared to their counterparts (Bugis *et al.,* 2025). The finding illustrates how BMI and anemia can jointly worsen disease outcomes, reinforcing the importance nutritional and hematological factors in the clinical assessment of patients with SCA.

Paradoxically, individuals with a high BMI (overweight or obese) are also at risk for iron deficiency and anemia. This is often due to a condition known as ‘’functional iron deficiency’’ where iron stores are adequate or even elevated, but the iron is not readily available for use by the body. The phenomenon is primarily driven by chronic inflammation associated with obesity, which increases the production of hepcidin, a hormone that inhibits iron absorption and traps iron in storage sites. Consequently, despite having sufficient iron stores, obese individuals may exhibit signs of iron deficiency anemia (WHO, 2021).

Chronic inflammation in obese individuals leads to elevated levels of hepcidin, reducing intestinal iron absorption and promoting iron sequestration within macrophages. A study published in the Journal of Nutrition found that higher BMI was associated with increased serum hepcidin levels and lower serum iron levels, reinforcing the connection between obesity, inflammation, and impaired iron metabolism (Qui *et al.,* 2022). Health practitioners should consider BMI when evaluating iron indices to tailor appropriate treatment strategies for both underweight and overweight patients. If obesity increase iron deficiency, it implies that it exposes sickle cell anemia patient to worsening anemia and hence poor health outcome.

Conversely, iron deficiency can occur due to poor nutritional status or increased demand, potentially affecting thrombotic risk through altered platelet function. Sickle red blood cells modulate clotting mechanism by entrapping red blood cells in a blood clot (thrombosis) (Kato, 2019).

Elevated BMI is strongly associated with an increased risk of thrombosis. Obesity leads to hypercoagulable state and increased secretion of plasminogen activator inhibitor (PAI)-1 and thrombin activatable fibrinolysis inhibitor (TAFI) which increases the likelihood of blood clot formation (Vilahur *et al.,* 2017). This is due to several factors, including increased levels of pro- coagulant factors, decreased fibrinolytic activity, low grade inflammation, increased oxidative stress and endothelial dysfunction as well as disturbance of lipids and glucose tolerance.

Adipose tissue, particularly visceral fat, secretes inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), which can promote thrombogenesis (Blokhin and Lentz, 2013).

Hyperlipidemia, elevated levels of triglycerides and low-density lipoprotein (LDL) cholesterol in obese individuals contribute to atherogenesis, which can further increase the risk of arterial thrombosis in sickle cell anemia patients.

Iron indices influences the risk of thrombosis. Both iron deficiency and iron overload have been implicated in thrombotic events, though the mechanisms may differ.

Iron Overload: Excessive iron can catalyze the formation of reactive oxygen species (ROS), leading to oxidative stress and endothelial injury which promotes thrombosis (Wun and Brunson, 2016).

Another study carried on sickle cell anemia patients recorded lower serum iron, lower mean cell volume and mean cell hemoglobin but within normal ferritin levels along

with elevated white blood cell and platelet counts compared to healthy individuals (Kamar *et al.,* 2024).

Iron indices is influenced by socio-demographic characteristics that include both age and gender

(Lal, 2020). As human continue to grow, several physiological stages create evident susceptibility to iron deficiency and overload. For instance, children and adolescents experience increased iron requirements due to growth and pubertal development, while women of reproductive age are particularly susceptible to iron deficiency anemia as a result of menstruation, pregnancy, and lactation (Burns *et al.,* 2025).

In contrast, adult men and postmenopausal women generally exhibit more stable iron stores, though chronic disease conditions may alter this balance. These age- and gender-specific variations are important in populations with chronic illnesses, such as sickle cell anemia, where the interaction between iron indices and body composition can significantly affect health outcomes.

Biologically, women of reproductive age have a higher risk of developing iron deficiency (ID).

Due to increased physiologic demand for iron required during menstruation and pregnancy as stated earlier. Although pregnant women stand at a higher risk of developing iron deficiency, about 52% experiencing ID globally (Means, 2020; Burns *et al.,* 2025). Untreated, iron deficiency can develop into iron deficiency anemia (IDA), which affects one in three women between the ages of 15–49 years worldwide

(Burns *et al.,* 2025). Consequently, women with elevated BMI face a compounded risk due to the dual burden of heavy menstrual blood loss and inflammation-driven iron sequestration (Burns *et al.,* 2025).

Age, another socio-demographic variable has also been shown to influence BMI among SCA.

Adolescents especially males are particularly vulnerable to growth delays. Rapid growth and high BMI adolescents are more prone to functional iron deficiency, whereas in older adults, excess adiposity combined with reduced physical activity may exacerbate systemic inflammation, thereby worsening iron bioavailability. From birth until adolescence, iron requirements increase, but are similar for both males and females, to support rapid physical and cognitive development. BMI plays a significant role in modulating iron indices across these different age groups and between genders. Higher BMI, particularly in overweight and obese individuals, has been consistently associated with reduced iron absorption and increased risk of iron deficiency.

Deficiency in micronutrients has raised several health complications globally, with a high rate of morbidity and mortality linked to the absence of iron in diet, thereby increasing the risk of anemia (Jamali *et al.,* 2021). Patients with sickle cell disease require higher energy and protein intake compared to healthy individuals. These patients tend to suffer from under nutrition if their energy intake is consistently low. Nutrients play a major role in the progression of and severity of SCA. SCA are frequently in a state of hypermetabolism due to the need to replace hemolysed red blood cells. Therefore, can lead to a very high demand for essential nutrients like iron,

folate, and vitamins. SCA patients with chronic hemolysis become vulnerable to both iron deficiency and iron overload, resulting from the alteration of iron homeostasis.

Although only little research exists that demonstrated the use of dietary interventions to tackle sickle cell anemia, however it is well established that unbalanced nutrition is a significant risk factor that adversely impacts clinical events, welfare, and vital processes for patients with SCA (Reber *et al.,* 2019). This is because there are no cost-effective methods to tackling SCA. Therefore efforts are being directed towards nutritional interventions to reduce ill health and improve the quality of life for SCA patients. Growth retardation from SCA is complex and may be due to the child's nutritional status, hematological and cardiovascular state, social factors, metabolic, and or altered endocrine function (Ukoha *et al.,* 2020).

Studies have shown that malnutrition, reflected by low BMI and stunting, is common among children and adolescents with SCA, particularly in low-resource settings where dietary diversity is poor. The role of malnutrition as one of the complications of SCA and the possible benefits of regular micronutrient supplements have been demonstrated in several studies (Reber *et al.,* 2019; Dike *et al.,* 2023). The interactions between nutritional status and heightened metabolic demands can influence disease severity, impair growth, and worsen quality of life in SCA patients. Nutritional interventions that balance adequate iron intake with monitoring for iron overload are therefore essential in managing the disorder.

Socioeconomic and Environmental Influences on BMI and Iron indices. Socioeconomic and environmental factors are two major factors that influence BMI

and iron indices in patients with SCA. Limited access to foods rich in nutrients, poor living conditions, level of education, social class, and health inequalities further influence nutrition and iron imbalance. In low- and middle-income countries like Nigeria. Household poverty and food insecurity have been proven to be associated with lower BMI and micronutrient deficiencies among children with SCA. In such circumstances, iron deficiency due to dietary inadequacies negatively impacts BMI. These disparities often occur as a result of unequal access to healthcare, low purchasing power, delay in diagnosis, and consumption of poor nutrient diets. Higher socioeconomic status and better education often correlate with healthier diets and increased physical activity, leading to better nutritional status. Environmental factors like neighborhood disorder and lack of green spaces for physical activity are linked to higher obesity rates and poorer diets, affecting iron deficiency and anemia.

Jesus *et al., (*2018) conducted a systematic review on socioeconomic and nutritional factors among children and adolescents with sickle cell anemia. The study found that adherence to treatment and attendance at follow-up visits especially for patients within the low socioeconomic class were often hindered by low household income, particularly when families live far from specialized healthcare facilities. The challenge was even more pronounced in children with sickle cell anemia (SCA), who experienced chronic disease and dependency.

However, in urban settings where transfusion services are available, iron overload becomes more prevalent, especially when chelation therapy (a form of treatment to improve survival rates) is inaccessible or unaffordable, which further complicate

overall health outcomes (Macharia *et al.,* 2022; Obeagu, 2025). This can create a dual burden where disadvantaged SCA patients may suffer from malnutrition and underweight caused by deficiency states. Hence, those with better access to transfusions may suffer from iron overload, both impairing BMI through different pathways. Urban-rural differences have been documented, with urban children more likely to have better BMI-for-age indices and iron indices than those in rural settings, largely due to differences in dietary diversity and healthcare access (Wake *et al.,* 2023).

Environmental and socioeconomic factors play a crucial role in shaping both BMI and iron indices among individuals with sickle cell anemia (SCA). Environmental exposures influence complex disease outcomes, and factors such as diet, nutritional supplementation and pollution. Access to health-promoting resources significantly contribute to variations in anemia prevalence and weight outcomes. For instance, environmental pollutants have been reported to disrupt iron homeostasis, while limited access to nutritious foods and safe spaces for physical activity fosters unhealthy eating patterns and sedentary lifestyles, thereby increasing the risk of obesity and elevated BMI, particularly in disadvantaged communities (Verde *et al.,* 2023).

Household living conditions also directly affect iron indices. Improved sanitation facilities and access to safe drinking water reduce the risk of infections and parasitic infestations, which otherwise deplete iron reserves and exacerbate anemia in SCA patients (Sahiledengle, Mwanri and Agho, 2024). Conversely, exposure to infectious

agents in resource-constrained environments increases iron requirements and heightens vulnerability to iron deficiency anemia. Food environments further shape nutritional status. Populations living in low-income neighborhoods are often surrounded by calorie-dense, affordable, but nutrient-poor foods, which promote energy imbalance and raise BMI (Zhang *et al.,* 2025). In contrast, individuals from higher socioeconomic backgrounds typically have better access to diverse, nutrient-rich diets and healthcare resources that mitigate the risk of anemia and poor nutritional outcomes.

Moreover, environmental stressors such as neighborhood disorder, crime, and deprivation are associated with higher rates of obesity, particularly among lower socioeconomic groups who lack access to preventive healthcare and structured interventions (Suglia *et al.,* 2016). Emerging evidence also links exposure to certain pollutants with obesity development, although the biological mechanisms remain under investigation (Amon, Kek and Klun, 2024).

CHAPTER THREE

MATERIALS AND METHODS

3.1 Study Area

This study was carried out at the Lagos University Teaching Hospital (LUTH), Idi-Araba, Lagos, Nigeria. Lagos is located in the South Western part of the country with an estimated population of over 21 million in 2016 by the National Population Commission of Nigeria that constitute over 6.4% of the national population of approximately 140 million as stated in 2006 National Population Census.

LUTH is a foremost tertiary hospital, established in 1961. It is located in Idi-Araba, Surulere, Lagos, Nigeria. LUTH is a 950 bedded tertiary hospital with referrals from all levels of health care delivery system within the metropolis and the surrounding environs. The hospital runs a weekly adult sickle cell clinic in the departments of medicine and hematology outpatient clinic with a combined attendance of approximately 20 sickle cell disease adult patients per week.

The Sickle Cell Foundation, Nigeria (SCFN), a non- governmental and non- profit making organization that ensures the proper care and control of sickle cell disorder in Nigeria. Hemoglobin SS were mostly referred from the SCFN that have about 40 registered patients. The SCFN is opposite the tertiary hospital (LUTH) in Idi-Araba.

3.2 Study Population

The study involved patients with sickle cell anaemia (HBSS) aged 18 years and above. The control group was made up of apparently healthy adults with haemoglobin AA phenotype matched for age and gender.

3.3 Study Design

This was a cross-sectional analytical study to determine the BMI and iron indices in patients with sickle cell anaemia and their association with disease severity.

3.4 Eligibility Criteria

3.4.1 Inclusion criteria

Sickle cell anemia patients who are 18 years and above.

Patients that were on steady state.

Those who gave informed consent.

3.4.2 Exclusion criteria

Patients with open wounds, patients with known bleeding disorders, or on iron supplements.

Pregnant women.

3.5 Sample Size Determination

The minimum sample size was determined using a formular designed to demonstrate significant difference in the comparison of two means (Kirkwood and Sterne, 2003). The minimum sample size was determined for each variable studied and the largest sample size which addresses all the objectives was selected. The formular is represented below:

$$n = (U+V)^2(\sigma_1^2 + \sigma_0^2) \Big/ (\mu_1 - \mu_0)^2$$

Where n is the calculated sample size and U is the one-sided percentage point of the nominal distribution corresponding to 100% minus the power of the study.

The power used in this study is 90% therefore u=1.28 (Kirkwood and Sterne, 2003)

V is the percentage of the normal distribution corresponding to the two-sided significance level. The significance level chosen for the study is 5%; therefore V=1.96. (Kirkwood and Sterne, 2003)

μ_0= mean of serum Ferritin in Healthy adults= 118.89 ng/ml (Moghadam *et al*., 2013).

μ_1= mean of serum ferritin in patients with CKD= 463.56 ng/ml (Kamar *et al*., 2024).

σ_1= standard deviation of serum level of ferritin in healthy adults=70.84ng/ml (Moghadam *et al*., 2013).

σ_2= standard deviation of serum level of ferritin in patients with CKD=655.05mg/ml (Kamar *et al*., 2024).

The calculated minimum sample size was 38.4. Hence a total of 45 patients was recruited for the study. Forty-five patients with HBSS and 45 controls with HB AA were recruited into this study.

3.6 Ethical Approval

Ethical approval with assigned number **ADM//DSCST/HREC/APP/6855** was obtained from Luth Health Research Committee (HREC) with Registration Number: NHREC: 19/12/2008a.

3.7 Sampling Technique

Stratified random sampling method was used with a sampling interval of three. The first patient was selected via balloting of first three patients that come in each day.

Every subsequent third patient who gave consent was recruited until the attainment of sample size.

3.8 Data Collection Tools

An interviewer structured questionnaire was used to collect data on sociodemographic, pains score and disease severity in all subjects (cases) recruited for the study. Other relevant data were extracted from patient's case note and routine physical examination were carried out. Pain was assessed using Adult Sickle Cell Quality of Life Measurement Information System -pain impact form (ASCQ-ME; for patients 18 years of age and older). This is a Patient-reported outcome tool. It is multidimensional and allow patients to quantitate the impact of pain on daily functioning and behavior (ASCQ-ME, 2017). Disease severity was assessed using Modified disease severity scoring system for sickle cell anemia (Hedo *et al.,* 1993; Akinyanju *et al*.,1989, 2005).

Anthropometric variables were measured according to Standard Anthropometric Assessment (ISAK, 2011). Stretch-stature method was used for weight and height measurement. A platform-type scale (Filozola) was used with a maximum capacity of 150kg and a precision of 0.1kg. Weight was determined without shoes and with light clothes. The participant stood on the center of the platform without support and with the weight distributed evenly on both feet. The weight was recorded to the resolution of the scale (nearest 0.1kg).

A portable stadiometer with an accuracy of 0.1cm was used for height measurement. The height vertical distance was measured from the crown of the head to the bottom of the feet (heels). The participant stands with bare feet together and the heels,

buttocks and upper part of the back touching the scale. Head was kept in Frankfurt plane with the help of a plastic ruler. The topmost point of the vertex was identified on the meter rule. Measurement was taken at the end of a deep inward breath.

Body Mass Index (BMI) were calculated by dividing the participant's weight (kg) with the height squared (m^2). Interpretation was done according to the criteria on Global Database on BMI recommended by the World Health Organization in 2021. Underweight is <18.5kg/m^2 normal weight is between 18.50 -24.99kg/m^2 and overweight is > and = 25.00kg/m^2.

3.9 Collection of Venous Blood

A total volume of 8ml of venous blood sample was collected from each participant using vacutainer blood collection procedure under aseptic conditions, with the observation of the universal safety precautions. The needle was inserted into the vein. Ensured that blood was in the needle's tubing, vacutainer tube was attached to the needle holder, and gently pushed the tube in to puncture the stopper, the vaccum withdraw blood into the tube. 3ml was put in a labeled di-potassium salt EDTA (K2-EDTA) for full blood count, 5ml into serum separating tube (SST) vacutainer for serum iron, ferritn and TIBC determination. All samples were collected in the morning during the clinic hours on the clinic days.

Full blood count was processed immediately (4-6hrs) using hematological auto-analyzer. Serum was gotten by centrifugation and stored at -20^oC till all samples are collected before analysis. Analysis was done using ELISA methods for serum ferritin and serum iron. TIBC was done with (SP 8001) spectrophotometer.

3.10 Laboratory Method

3.10.1 Full Blood Count (FBC)

FBC include packed cell volume, red blood cell, total white blood cell count, and platelet count. Full blood count was done using auto-analyzer, Mindray BC 3200, RN-13000934 manufactured in China in 2011. The assay protocol was adhered to according to manufacturer's instruction.

Principle: Full blood count principle is based on Coulter Principle which detects and measures the changes in electrical impedance produced by a cell or particle suspended in a conductive liquid (diluent) transversing through a small aperture, allowing it to differentiate between RBCs, WBCs and PLTs (Genc *et al*., 2017) . The Coulter Principle offers the sensitivity, accuracy and flexibility needed for studying cell distribution in organism with different biological characteristics.

To ensure that the blood cells are evenly distributed, blood sample shaker (Roll Mixer, Wincom) made in China was used to agitate the sample. The cells are suspended in a fluid stream and their properties are measured as they flow past sensors in a flow cytometry. Automated analyzer are regularly calibrated, the analyzer uses internal controls and standards to verify that its results are correct.

Full blood count Reference Range

Red Blood Cell: Hemoglobin - male: 13.2-16.6g/dl; female: 11.6-15g/dl

Hematocrit - male: 38.3- 48.6%; female: 35.5-44.9%

White Blood Cell: 3.4 - 9.6 x 10^9/L

Platelet – male: 135 - 371x10^9/L; female: 157 – 371 x 10^9/L.

3.10.2 Estimation of Serum Ferritin

Serum ferritin levels were measured quantitatively using a commercially available Enzyme-Linked Immunosorbent Assay (ELISA) kit produced by Accubind, USA. The manufacturer's instructions were strickly followed.

Principle: The assay relies on antibodies to detect a target antigen in the sample resulting in antibody-antigen complex and signal detection. The lower the amount of antigen in the sample, the stronger the signal due to more labeled antigen in the well. The linked enzyme acting on the substrate produced a colour change which can be measured photometrically using micro reader. Sandwich Elisa was used for ferritin analysis because of its high sensitivity and specificity in detecting the presence of small antigen in a sample.

Before proceeding with the assay, the reagents, controls and specimen were brought to room temperature (20-27^{o}C).

i. Application of 25ul of ferritin calibrator, participant specimen and control were pipetted into the respective well.
ii. Addition of 100ul of the ferritin biotin reagent was made closely to the bottom of each well.
iii. Mixed properly by swirling the microplate gently for 20-30 seconds and covered.

iv. Incubation was made at room temperature for 30 minutes, followed by 3 times washing using wash buffer in automatic plate washer to remove unbound substances and reagents.

v. Absorber paper was used to blot dry the plate.

vi. Hundred microliter of the ferritin enzyme conjugate was added to each well carefully.

vii. Incubated for 30 minutes at room temperature, used automatic plate washer to wash again to ensure accurate and reliable results by removing contaminants as much as possible.

viii. Hundred microliter of substrate solution was added to all wells carefully, incubated for 15 minutes at room temperature after which 50ul of stop solution was made and mixed gently for 15-20 second.

ix. Results were read within 30 minutes of reaction stoppage, at wavelength of 630nm using a microplate reader. The concentration of serum ferritn were also determined by plotting the absorbance values against a standard curve and expressed in ng/ml.

Ferritin reference range in adult 15- 300ng/ml.

Men: 12-300ng/ml; Female: 10-150ng/ml

3.10.3 Serum Iron Determination

Serum gotten from serum separating tube was used for iron analysis using the Mindray kit on Mindray BS-240 autoanalyzer and it comprises of R1 and R2. R1 is Acetate buffer with reducing agent and R2 is Ferrozine.

Principle: In acidic conditions, iron was liberated from transferrin and reduced the released Fe^{3+} to Fe^{2+} ions. Ferrous ions then react with ferrozine to form a colored complex. The color intensity is directly proportional to the concentration of the iron content in the sample which is measured photometrically.

Procedure for Estimation of Serum Iron

Before proceeding with the assay, the reagents, controls and samples were brought to room temperature (20-27^{o}C).

- Two hundred and fifty microliter of R1 were pipetted into suitable cuvette in a cuvette holder.
- Addition of 20ul of calibrator, control and non hemolysed or lipemic serum from SST (samples) was made respectively (A1).
- Mixed incubated at 37^{o}C for 5-10 minute
- Read the absorbance of A1 at 578nm then 250ul of R2 was added (A2).
- Mixed thoroughly with the aid of mixer and incubated at 37^{o}C for 5 minutes.
- The absorbance of A2 was read at 578nm photometrically.

Then, the Absorbance, A=A2-A1

The Mindray System BS-240 automatically calculated the results on the absorbance difference between the sample and the standard calibrator. So the method used was endpoint. Strict adherence to the manufacturer's instructions was observed.

Iron reference ranges in healthy adults: Male 80-180ug/dl; Female 60-160ug/dl.

3.10.4 Estimation of Total Iron Binding Capacity (TIBC)

Principle: The TIBC test estimation using Biosystems kit was based on addition of excess iron to the sample to saturate transferrin. The unbound iron was removed by adsorption using magnesium hydroxide carbonate and iron bonded to transferrin was then measured spectrophotometrically at 570nm. Adherence to manufacturer's instruction was ensured.

Before commencing with the assay, all reagents, controls, standard calibrator and specimens were brought to room temperature (20 – 27^{o}C).

- Five hundred microliter of standard calibrator, non hemolysed participant samples and control were pipetted into respective tubes, addition of 1000ul of iron reagent (iron chloride (111)) was made so as to saturate the available iron binding sites on transferrin.
- Mixed thoroughly and incubated at 5-30 minute at room temperature, then one spoonful of magnesium hydroxide carbonate was added and mixed while allowing to stand for 30-60 minute at room temperature, mixing thoroughly several times for unbound excess iron to be removed.
- Centrifuged at 3000rpm for 10 minute to sediment the precipitate and the clear supernatant was collected for measurement using the kit iron ferrozine.
- The absorbance was read at 570nm using spectrophotometer against a blank.
- The absorbance of the sample was compared with that of the standard to determine iron concentration (TIBC).

Refrence range for adult TIBC is 240 - 450ug/dl

.3.10.5 Assessment of Disease Severity

The clinical severity of sickle cell anemia was determined using the Yes/No clinical severity scoring system developed by (Hedo *et al.,*1993; Akinyanju *et al.,* 1989, 2005**)**. This was obtained by summing up the number of crises per year (0-1=0, 2-3=1, ≥ 4 = 2), number of complications and degree of anemia. Each clinical feature, including vaso-occlusive crises, sequestration, acute chest syndrome, osteomyelitis, renal failure, heart failure, avascular necrosis of the femoral head, pneumonia, pigment gallstone jaundice and dehydration (was scored as **"Yes = 1"** if present or **"No = 0"** if absent. The total score for each participant was obtained by summing all "Yes" responses. Based on the total scores, disease severity was categorized as mild (0–3 points), moderate (4–7 points), or severe (> 7 points).

3.11 Data Analysis

Data obtained from the questionnaires and results from sample analysis was entered into Microsoft Excel 2026 spreadsheet and cleaned, then exported into and analysed using a Graphpad Prism version 09 for analysis. Results were presented using tables, pie charts as appropriate. Normality were determined for continuous variables using the Kolmogorov-Smirnor Test. The continuous variables were presented as mean and standard deviation for parametric variables or median and interquartile range for non parametric variables. Statistical differences between means for parametric continuous variables was tested using the Independent T test and Mann Whitney test for non - parametric test for several independent samples as applicable. Categorical variables

were presented as frequency and percentages. Spearman's rho correlation coefficient was used to determine the correlation between two variables. Two –tailed P- value of less than 0.05 were considered significant.

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CHAPTER FOUR

RESULTS

A total of 90 participants were included in the study, divided equally between the Sickle Cell Anaemia (SCA) group and the control group. The demographic characteristics of the participants are summarized below. Females made up the majority of the study population. In the SCA group, 32 participants (50.8%) were female, similar to 31 females (49.2%) in the control group. Males represented 28.9% of the SCA group and 31.1% of the controls, giving an overall distribution of 30% males and 70% females. Most participants were young adults. Nearly half of the SCA group (46.7%) and 40% of the control group were between 18 and 25 years old. Those aged 26–35 made up 20% of the SCA group and 17.8% of the controls. Participants aged 36–45 accounted for 22.2% of the SCA group and 28.9% of the controls. Smaller proportions fell within the 46–55 age bracket (8.9% in both groups), while only a few participants were aged 56 and above (2.2% in SCA and 4.4% in controls). Overall, the age distribution shows a predominantly youthful population. Christianity was the major religion among participants, with 82.2% represented in both the SCA and control groups. Islam accounted for 17.8% in each group. A considerable proportion of the SCA group (48.9%) were self-employed, compared with only 11.1% in the control group. Conversely, civil servants were more common among controls (53.3%) than in the SCA group (11.1%). Students formed a consistent share of both groups—35.6% in SCA and 33.3% in controls. Only a few participants worked in private-sector jobs. Most participants identified as Yoruba, comprising 73.3% of the SCA group and 62.2% of the controls. Igbo participants accounted for

20% of the SCA group and 33.3% of the controls. A small proportion belonged to other ethnic groups. There were more single participants overall. In the SCA group, 84.4% were single compared with 55.6% in the control group. Married individuals made up 15.6% of the SCA group and 44.4% of the controls. Most participants had tertiary education77.8% in the SCA group and 71.1% among controls. Those with only secondary education represented 22.2% of the SCA group and 28.9% of the control group.

Table 4.2 compares the red blood cell indices of case and control subjects. The results reveal that hemoglobin (HGB), packed cell volume (PCV), and red blood cell count (RBC) were all significantly lower in the case group compared to the control group ($p < .001$). In contrast, mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) were significantly higher in the case subjects ($p < .001$), while the mean corpuscular hemoglobin concentration (MCHC) showed no significant difference ($p = .463$). Additionally, both red cell distribution width (RDW-CV and RDW-SD) were higher in the case group compare to control group ($p < .001$).

Table 4.3 shows the white blood cell (WBC) distribution between case and control groups. The mean total WBC count was significantly higher in the case group ($p < .001$). However, the differential counts including lymphocytes, granulocytes, and mid cells showed no significant differences ($p > .05$).

Table 4.4 presents the distribution of platelet indices between case and control groups. The total platelet count (PLT) and plateletcrit (PCT) were significantly higher among

the SCA subjects ($p < .001$). On the other hand, mean platelet volume (MPV) and platelet distribution width (PDW) did not differ significantly between groups ($p > .05$).

Table 4.5 compares iron indices indicators between SCA and healthy control subjects. The results showed a significant increase in ferritin concentration among the cases ($p < .001$) and a significant decrease in total iron-binding capacity (TIBC) ($p < .001$). Serum iron levels, however, did not differ significantly ($p > 0.05$).

Table 4.6 presents the distribution of body mass index (BMI) categories among the study groups. Most participants in both groups fell within the normal weight range (18.5–24.9), accounting for 28 of SCA and 36 of the healthy controls. However, a significantly higher number of test subjects were underweight (13 versus 1 in the control group; $p = 0.001$), while fewer were overweight (4 compared to 8 controls). No obese participants were recorded in either group.

Table 4.7 showed the Mean Comparison of Red Blood Cell Indices by BMI Category in Test and Control Subjects. Across all BMI categories (underweight, normal weight, and overweight), the mean hemoglobin (HGB), packed cell volume (PCV), and red blood cell count (RBC) were significantly lower in the test subjects compared with the healthy control ($p < 0.05$). For mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH), the test subjects recorded significantly higher values than the controls among normal weight and overweight individuals ($p < 0.05$), while no significant difference was observed in the underweight category. Mean corpuscular hemoglobin concentration (MCHC) did not differ significantly between test and control groups across all BMI categories ($p > 0.05$). In contrast, both red blood cell

distribution width (RDW-CV) and red blood cell distribution width–standard deviation (RDW-SD) were significantly higher in the test subjects across all BMI categories ($p < 0.05$).

Table 4.8: showed the Mean Comparison of White Blood Cell Distribution by BMI Category in SCA and Healthy Controls. White blood cell counts (WBC) were significantly higher in SCA subjects than in controls across all BMI categories ($p < 0.05$). Lymphocytes were higher in cases than controls across all BMI groups ($p < 0.001$), whereas granulocytes were significantly lower ($p < 0.001$). The proportion of mid – cell percentage (Mid%) was also significantly increased in the case group across all BMI categories ($p < 0.01$).

Table 4.9 showed the Mean Comparison of Platelet Parameters by BMI Category in Test and Control Subjects. Platelet counts (PLT) were significantly higher in test subjects than in controls across all BMI categories ($p < 0.01$). Mean platelet volume (MPV) and platelet distribution width (PDW) did not differ significantly between test and control subjects ($p > 0.05$). Plateletcrit (PCT) values were significantly higher in the test group across all BMI categories ($p < 0.01$).

Table 4.10 showed the Mean Comparison of Iron indices Parameters by BMI Category in SCA and Healthy Controls. Ferritin levels were significantly higher in sickle cell anemia subjects compared with controls across all BMI categories ($p < 0.001$). Serum iron concentrations showed no significant difference between test and control groups ($p > 0.05$) across all BMI categories. Total iron-binding capacity

(TIBC) was significantly lower in sickle cell anemia subjects compared with controls ($p < 0.001$).

Table 4.11 shows the disease severity among sickle cell anemia subjects. The disease severity analysis among sickle cell anemia subjects reveals that painful crises occurred most commonly on a yearly basis (60%), followed by quarterly (22.2%) and bimonthly (11.1%) frequencies. The majority of participants experienced four crisis per year (55.6%), while 37.8% experienced two annually. In terms of crisis types, acute chest syndrome (50%), avascular necrosis (48.9%), vaso-occlusive crisis (48.9%), and osteomyelitis (47.8%) were the most prevalent complications. Renal failure (1.1%) and heart failure (0%) were rarely observed among participants. For anemia grading, mild to moderate anemia (hemoglobin>=8<10g/dl and >=6<8g/dl) predominated, affecting 75.6% of patients, while only 6.7% presented with severe anemia (hemoglobin>=4<6g/dl).

Table 4.1: Demographic characteristics of the study population in the Study Area

Demographic	SCA Freq. (%)	Controls Freq. (%)	Total Freq. (%)
Sex			
Male	13(28.9)	14(31.1)	27(30.0)
Female	32(50.8)	31(49.2)	63(70.0)
Age			
18–25	21 (46.7%)	18 (40.0%)	39 (43.3%)
26–35	9 (20.0%)	8 (17.8%)	17 (18.9%)
36–45	10 (22.2%)	13 (28.9%)	23 (25.6%)
46–55	4 (8.9%)	4 (8.9%)	8 (8.9%)
56+	1 (2.2%)	2 (4.4%)	3 (3.3%)
Religion			
Christian	37(82.2)	37(82.2)	74(37(82.2)
Islam	8(17.8)	8(17.8)	16(17.8)
Occupation			
Student	16(35.6)	15(33.3)	31(32.4)
Self employed	22(48.9)	5(11.1)	27(30.0)
Civil servant	5(11.1)	24(53.3)	29(32.2)
Private	2(2/2)	1(1.1)	3(3.3)
Ethnicity			
Yoruba	33(73.3)	28(62.2)	61(67.8)
Igbo	9(20.0)	15(33.3)	24(26.7)
Others	3(6.7)	2(4.4)	5(55.6)
Marital status			
Single	38(84.4)	25(55.6)	63(70.0)
Married	7(15.6)	20(44.4)	27(30.0)
Education			
Secondary	10(22.2)	13(28.9)	23(25.6)
Tertiary	35(77.8)	32(71.1)	67(74.4)

Table 4.2: Mean levels of hemoglobin and red blood cell indices compared between SCA subjects and healthy controls

Parameters	Case (Mean ± SD) (n = 45)	Control (Mean ± SD) (n = 45)	t-Value	P-Value
HGB (g/dL)	8.39 ± 1.53	12.42 ± 1.60	-19.27	.000
PCV (%)	25.66 ± 5.18	39.49 ± 4.98	-19.11	.000
RBC ($\times 10^{12}$/L)	2.87 ± 0.67	4.66 ± 0.59	-17.83	.000
MCV (FL)	93.01 ± 13.09	85.54 ± 6.79	3.86	.000
MCH (pg)	29.27 ± 4.26	26.78 ± 2.24	3.65	.001
MCHC (g/dL)	31.95 ± 0.83	31.80 ± 1.35	0.74	.463
RDW-CV (%)	17.67 ± 3.64	13.42 ± 1.70	8.37	.000
RDW-SD (FL)	59.00 ± 13.41	42.48 ± 3.72	8.29	.000

$P < 0.05$ indicate significant difference

Key

- **HGB (g/dL):** Hemoglobin concentration
- **PCV (%):** Packed Cell Volume
- **RBC ($\times 10^{12}$/L):** Red Blood Cell Count
- **MCV (FL):** Mean Corpuscular Volume
- **MCH (pg):** Mean Corpuscular Hemoglobin
- **MCHC (g/dL):** Mean Corpuscular Hemoglobin Concentration
- **RDW-CV (%):** Red Cell Distribution Width–Coefficient of Variation
- **RDW-SD (FL):** Red Cell Distribution Width–Standard Deviation

Table 4.3: Mean distribution of white blood cells among SCA subjects and healthy controls

Parameters	Case (Mean ± SD) (n = 45)	Control (Mean ± SD) (n = 45)	t-Value	P-Value
WBC	8.02 ± 3.28	5.73 ± 1.93	5.47	.000
Lymph %	46.17 ± 15.75	44.47 ± 14.22	0.64	.528
Gran %	44.83 ± 13.41	46.58 ± 14.50	–0.81	.423
Mid %	11.23 ± 4.77	10.41 ± 3.31	0.97	.336

$P < 0.05$ indicate significant difference

Keys

- **WBC:** White Blood Cell Count
- **Lymph (%):** Lymphocyte Percentage
- **Gran (%):** Granulocyte Percentage
- **Mid (%):** Mid-Cell Percentage (includes monocytes, eosinophils, and basophils)

Table 4.4: Mean levels of platelet parameters compared between SCA subjects and healthy controls

Parameters	Case (Mean ± SD) (n = 45)	Control (Mean ± SD) (n = 45)	t-Value	P-Value
PLT (×10⁹/L)	349.20 ± 145.22	235.36 ± 68.74	5.68	.000
MPV (FL)	8.82 ± 0.88	8.90 ± 0.64	-0.54	.591
PDW (FL)	15.29 ± 0.38	15.37 ± 0.34	-1.33	.190
PCT (%)	0.30 ± 0.11	0.21 ± 0.06	5.59	.000

$P < 0.05$ indicate significant difference

Keys

- **PLT (×10⁹/L):** Platelet Count
- **MPV (FL):** Mean Platelet Volume
- **PDW (FL):** Platelet Distribution Width
- **PCT (%):** Plateletcrit

Table 4.5: Mean levels of iron indices compared between study participants

Parameters	Case (Mean ± SD) (n = 45)	Control (Mean ± SD) (n = 45)	t-Value	P-Value
Ferritin	233.72 ± 144.39	72.55 ± 63.80	7.289	.000
Iron	104.28 ± 44.95	101.92 ± 25.75	0.313	.756
TIBC	118.54 ± 29.13	157.39 ± 35.50	-5.773	.000

$P < 0.05$ indicate significant difference

Key

TIBC: Total Iron-Binding Capacity

Table 4.6: Categorization of Body Mass Index among Participants

BMI Category	Case (n = 45)	Control (n = 45)	P values
Underweight (<18.5)	13	1	0.001*
Normal weight (18.5–24.9)	28	36	
Overweight (25.0–29.9)	4	8	
Obese (≥30)	0	0	

P < 0.05 indicate significant difference

Key

BMI: Body Mass Index

Table 4.7 BMI and mean comparison of hemoglobin and red blood cell indices in SCA and healthy controls

Parameters	BMI Category	Case (mean ± SD)	Control (mean ± SD)	p-value
HGB (g/dL)	Underweight	8.50 ± 1.82	12.60 ± 1.59	0.000
	Normal weight	8.24 ± 1.20	12.41 ± 1.30	0.000
	Overweight	8.22 ± 2.42	12.62 ± 1.82	0.029
PCV (%)	Underweight	26.66 ± 5.82	39.53 ± 5.24	0.000
	Normal weight	25.73 ± 3.75	39.00 ± 3.84	0.000
	Overweight	25.70 ± 8.13	40.45 ± 5.98	0.029
RBC ($\times 10^{12}$/L)	Underweight	2.92 ± 0.58	4.50 ± 0.58	0.000
	Normal weight	2.84 ± 0.63	4.60 ± 0.45	0.000
	Overweight	2.77 ± 0.88	4.91 ± 0.20	0.014
MCV (FL)	Underweight	91.46 ± 6.46	88.47 ± 3.98	0.156
	Normal weight	92.93 ± 13.35	85.36 ± 5.87	0.011
	Overweight	93.70 ± 4.45	81.67 ± 6.75	0.030
MCH (pg)	Underweight	29.07 ± 2.05	27.72 ± 2.09	0.097
	Normal weight	29.69 ± 4.55	26.63 ± 1.90	0.003
	Overweight	29.90 ± 2.33	25.48 ± 2.05	0.030
MCHC (g/dL)	Underweight	31.89 ± 0.67	31.76 ± 0.62	0.610
	Normal weight	31.99 ± 0.68	31.83 ± 1.46	0.618
	Overweight	32.17 ± 1.04	31.25 ± 0.45	0.176
RDW-CV (%)	Underweight	17.86 ± 2.61	13.79 ± 2.22	0.000
	Normal weight	17.08 ± 3.61	13.17 ± 0.97	0.000
	Overweight	17.05 ± 2.19	13.30 ± 0.96	0.033
RDW-SD (FL)	Underweight	57.39 ± 8.72	44.58 ± 5.70	0.000
	Normal weight	57.93 ± 11.83	41.51 ± 3.47	0.000
	Overweight	56.98 ± 4.47	40.02 ± 2.32	0.002

$P < 0.05$ indicate significant difference

Key

- **HGB (g/dL):** Hemoglobin concentration
- **PCV (%):** Packed Cell Volume
- **RBC (×10⁶/µL):** Red Blood Cell Count
- **MCV (FL):** Mean Corpuscular Volume
- **MCH (pg):** Mean Corpuscular Hemoglobin
- **MCHC (g/dL):** Mean Corpuscular Hemoglobin Concentration
- **RDW-CV (%):** Red Cell Distribution Width–Coefficient of Variation
- **RDW-SD (FL):** Red Cell Distribution Width–Standard Deviation
- **BMI:** Body Mass Index

Table 4.8. Comparison of the means of white blood cell (WBC) distribution and BMI in case and control participants.

Distribution	BMI Category	Case (Mean ± SD)	Control (Mean ± SD)	p-value
WBC (×10⁹/L)	Underweight	9.62 ± 3.25	6.11 ± 1.84	0.001
	Normal weight	9.83 ± 3.41	6.04 ± 1.76	0.000
	Overweight	10.05 ± 3.02	6.26 ± 1.85	0.002
Lymph (%)	Underweight	62.43 ± 11.30	40.21 ± 7.85	0.000
	Normal weight	63.72 ± 12.54	39.35 ± 6.93	0.000
	Overweight	64.15 ± 10.12	38.48 ± 6.56	0.001
Gran (%)	Underweight	30.22 ± 9.84	54.83 ± 8.51	0.000
	Normal weight	29.41 ± 8.97	55.41 ± 7.63	0.000
	Overweight	28.75 ± 7.45	56.12 ± 6.92	0.001
Mid (%)	Underweight	7.35 ± 1.62	4.96 ± 0.87	0.000
	Normal weight	6.87 ± 1.74	5.07 ± 1.04	0.002
	Overweight	7.10 ± 1.53	5.12 ± 0.91	0.004

$P < 0.05$ indicate significant difference

Keys

· **WBC:** White Blood Cell Count
· **Lymph (%):** Lymphocyte Percentage
· **Gran (%):** Granulocyte Percentage

· **Mid (%):** Mid-sized Cell Percentage (includes monocytes, eosinophils, and basophils)
BMI: Body Mass Index

Table 4.9 Mean Comparison of platelet parameters and BMI Category in SCA subjects and healthy controls

Parameters	BMI Category	SCA (Mean ± SD)	Control (Mean ± SD)	p-value
PLT (×10⁹/L)	Underweight	352.20 ± 145.31	235.12 ± 68.50	0.001
	Normal weight	348.80 ± 142.66	236.05 ± 69.20	0.000
	Overweight	343.90 ± 151.02	237.10 ± 67.85	0.002
MPV (FL)	Underweight	8.86 ± 0.92	8.93 ± 0.64	0.583
	Normal weight	8.80 ± 0.85	8.88 ± 0.62	0.605
	Overweight	8.79 ± 0.91	8.90 ± 0.65	0.598
PDW (FL)	Underweight	15.31 ± 0.36	15.38 ± 0.34	0.242
	Normal weight	15.28 ± 0.37	15.35 ± 0.33	0.270
	Overweight	15.25 ± 0.38	15.34 ± 0.35	0.295
PCT (%)	Underweight	0.30 ± 0.11	0.21 ± 0.06	0.001
	Normal weight	0.30 ± 0.10	0.21 ± 0.05	0.000
	Overweight	0.29 ± 0.12	0.21 ± 0.06	0.002

$P < 0.05$ indicate significant difference

Keys

- **PLT (×10⁹/L):** Platelet Count
- **MPV (FL):** Mean Platelet Volume
- **PDW (FL):** Platelet Distribution Width
- **PCT (%):** Plateletcrit
- **BMI:** Body Mass Index

Table 10. Comparison of BMI and iron indices in SCA subjects and healthy controls

Parameters	BMI Category	Case (Mean ± SD)	Control (Mean ± SD)	p-value
Ferritin (ng/mL)	Underweight	232.85 ± 142.64	72.31 ± 63.45	0.000
	Normal weight	234.11 ± 145.10	72.80 ± 64.10	0.000
	Overweight	235.62 ± 146.73	73.06 ± 63.90	0.001
Iron (µg/dL)	Underweight	104.10 ± 43.82	101.76 ± 25.43	0.751
	Normal weight	104.52 ± 44.96	101.89 ± 25.71	0.754
	Overweight	104.90 ± 45.21	102.11 ± 25.83	0.752
TIBC (µg/dL)	Underweight	118.46 ± 28.74	157.12 ± 35.28	0.000
	Normal weight	118.61 ± 29.04	157.44 ± 35.42	0.000
	Overweight	118.76 ± 29.25	157.61 ± 35.67	0.000

$P < 0.05$ indicate significant difference

Key

TIBC: Total Iron-Binding Capacity

BMI: Body Mass Index

Table 4.11: Disease severity scoring system for sickle cell anemia subjects

	Frequency	Percent
How often do you have painful crisis		
Monthly	3	6.7
Bimonthly	5	11.1
Quarterly	10	22.2
Yearly	27	60.0
Total	45	100.0
Type of crisis per year		
2	17	37.8
3	3	6.7
4	25	55.6
Total	45	100.0
Type of crisis per year		
VOC	44	48.9
Sequestration	0	0.0
Acute chest syndrome	45	50.0
Osteomyelitis	43	47.8
Renal failure	1	1.1
Heart failure	0	0.0
Avascular	44	48.9
Pneumonia	29	32.2
Pigment gallstone of femoral head	13	14.4
Dehydrated	43	47.8
Anemia		
.0	8	17.8
1	21	46.7
2	13	28.9
3	3	6.7
Total	45	100.0

Figure 4.1 shows the Hb levels in sickle cell anemia **(**SCA) subjects, reveals that majority of the participants experience some level of anemia, with 76% of patients falling into the mild to moderate categories (Hb >= 6 g/dl < 10 g/dl). Specifically, the largest group being the 47% subjects with mild anemia (Hb 8 g/dl < 10g/dl), followed by 29% with moderate anemia (Hb 6 g/dl < 8 g/dl). In addition, 9% of the population suffers from severe anemia (Hb 4 g/dl < 6g/dl), while only 15% maintains the highest hemoglobin levels (Hb >= 10g/dl).

Figure 4.2**:** Pie chart showing the overall grade of disease severity scoring system for sickle cell anemia patients This chart reveals that 9% of the participants had mild anemia, **64%** had moderate anemia, and 27% had severe anemia.

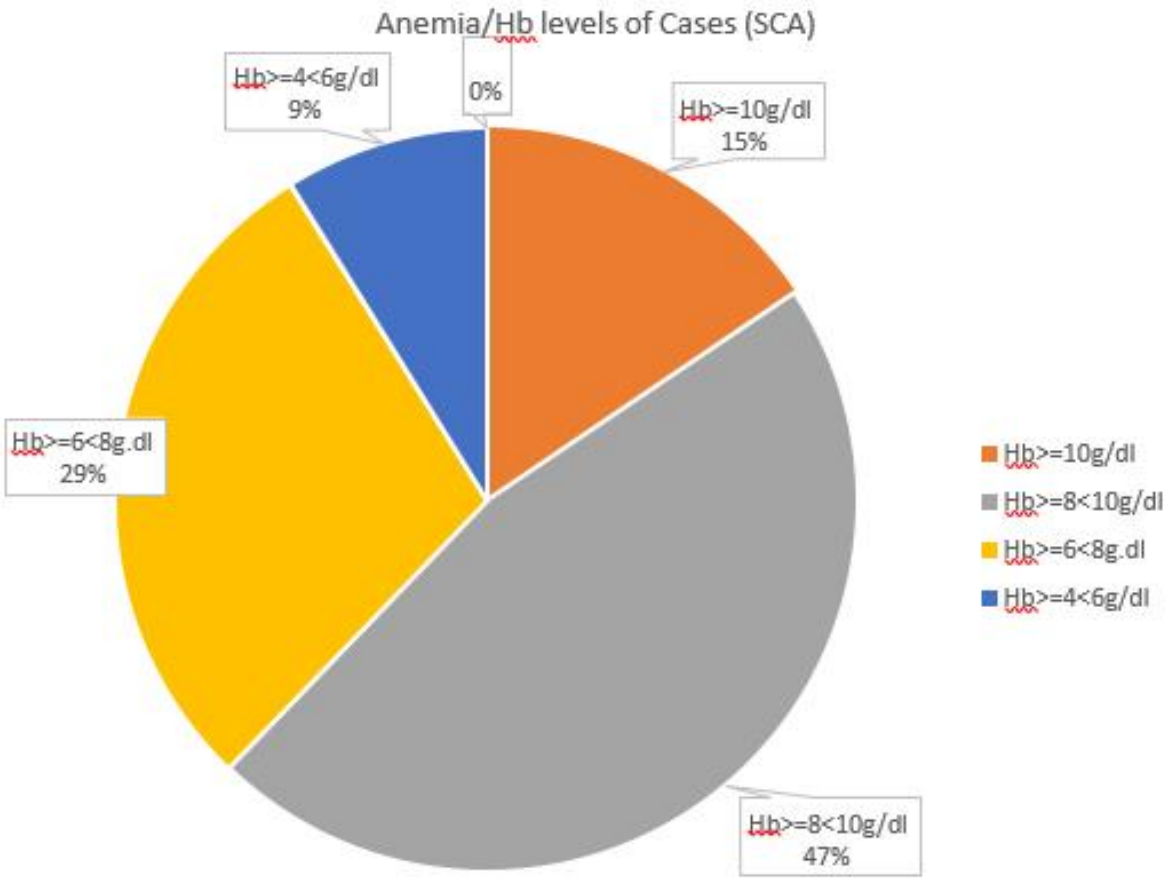

Figure 4.1: Anemia/Hg levels of Sickle Cell Anemia Subjects

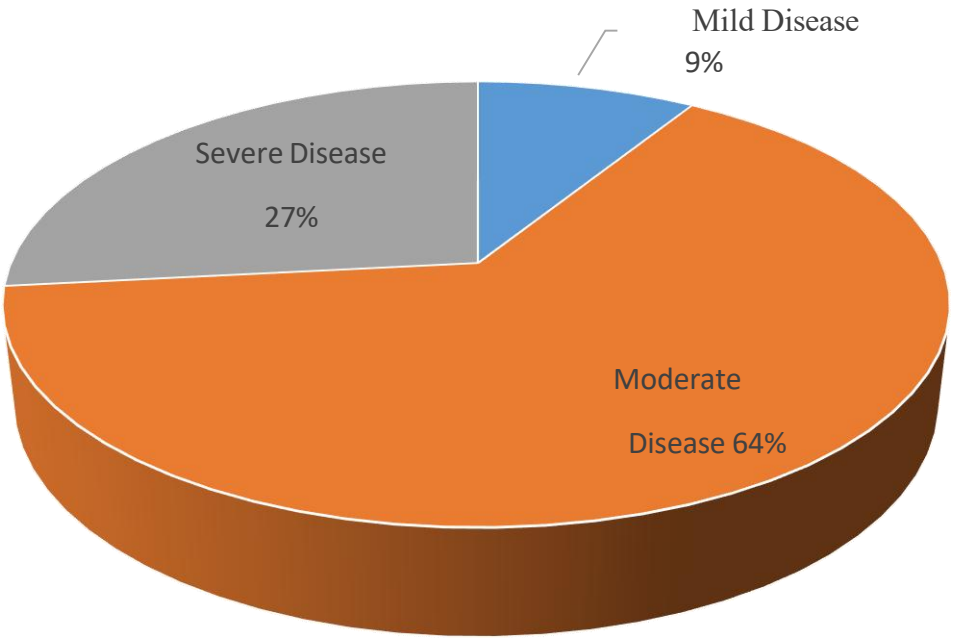

Figure 4.2: Pie chart showing overall grade of Disease Severity Scoring System for Sickle Cell Anemia Subjects.

Table 4.12 shows the distribution of types of crisis experienced by sickle cell anemia subjects. All the patients (100%) experienced vaso-occlusive crises (VOC) and acute chest syndrome, making them the most prevalent crisis. Osteomyelitis (97.8%), avascular necrosis (97.8%), and dehydration (95.6%) were also highly common among participants. In contrast, renal failure (2.2%) and heart failure (2.2%) were the least reported complications.

Table 4.13 shows the distribution of pain assessment responses among sickle cell anemia patients. A large proportion (91.1%) of participants reported experiencing pain "sometimes" and "often," with 46.7% stating that pain prevented them from performing daily activities. Additionally, 42.2% reported being "totally in pain", and nearly half (48.9%) experienced "sudden attacks of pain".

The Table 4.14 showed the correlation analysis that examined the relationships between body mass index (BMI), indicators of iron indices, and the severity of sickle cell anemia among the cases. The findings revealed that BMI showed no significant association with any of the severity indicators or iron parameters. This suggests that body composition, as measured by BMI, does not appear to influence either the iron indices or the clinical manifestations of sickle cell anemia in this sample.

With respect to iron indices, serum ferritin demonstrated weak and statistically insignificant correlations with the different measures of disease severity, including vaso-occlusive crisis (VOC), sequestration crisis, acute chest syndrome, osteomyelitis, and other complications. Although the correlation between ferritin and osteomyelitis was moderately positive, it did not reach statistical significance, implying that

elevated ferritin levels may reflect underlying inflammation or infection rather than a direct effect on disease severity.

Serum iron, however, showed more notable patterns. There was a significant negative correlation between serum iron and vaso-occlusive crisis ($r = –0.351$, $p < 0.05$), indicating that lower iron levels were associated with higher frequency or severity of crises. Similarly, serum iron exhibited a significant negative relationship with pneumonia ($r = –0.322$, $p < 0.05$), suggesting that iron deficiency may predispose patients to greater susceptibility to infections and acute complications. Other correlations involving serum iron, such as those with total iron-binding capacity (TIBC), BMI, and other clinical conditions, were weak and not statistically significant

Table 4.12: Types of crisis experienced by sickle cell anemia subjects in the Study Area

Type of Crisis	Frequency	Percentages
VOC	45	100.0
Sequestration	0	0.0
Acute chest syndrome	45	100.0
Osteomyelitis	44	97.8
Renal failure	1	2.2
Heart failure	1	2.2
Avascular necrosis of femoral head	44	97.8
Pneumonia	28	63.6
Pigment gallstone jaundice	20	44.4
Dehydrated	43	95.6

Table 4.13: Pain assessment report in sickle cell anemia subjects in the Study Area

In the past 7 days	Never Freq.(%)	Rarely Freq.(%)	Sometimes Freq.(%)	Often Freq.(%)	Always Freq.(%)
Pain so bad you could not do anything the whole day	18(40.0)	20(44.0)	7(15.6)	0(0.0)	0(0.0)
Totally in pain	1(2.2)	3(6.7)	15(33.3)	19(42.2)	7(15.6)
Sudden attack	7(15.6)	15(33.3)	22(48.9)	1(2.2)	0(0.0)
Pain so bad could not get out of bed	9(20.0)	22(48.9)	14(31.1)	0(0.0)	0(0.0)
Severe pain	3(6.8)	19(43.2)	18(40.9)	4(9.1)	0(0.0)
You cancelled plans	7(15.6)	14(31.1)	19(42.2)	5(11.1)	0(0.0)
You stopped what you were doing	1(2.2)	16(35.6)	24(53.3)	4(8.9)	0(0.0)
Difficult to finish what you were doing	6(13.3)	18(40.0)	19(42.2)	2(4.4)	0(0.0)
Terrified you might have a crisis	7(4.4)	6(28.9)	22(40.0)	6(20.0)	4(4.4)
How bad is the usual pain	2(4.5)	13(29.5)	18(40.9)	9(20.5)	2(44.5)
Bad pain in the joints	1(2.2)	12(26.7)	22(48.9)	5(11.1)	5(11.1)
Pain preventing you from doing anything	8(17.8)	14(31.1)	21(46.7)	2(4.4)	0(0.0)

Table 4.14 Correlation of Body Mass Index, Iron indices and the Severity of Sickle Cell Anemia

	FERRITIN		IRON		TIBC		BMI	
	Pearson Correlation	Sig. (2-tailed)	Pearson Correlation	Sig. (2-tailed)	Pearson Correlation	Sig. (2-tailed)	Pearson Correlation	Sig. (2-tailed)
VOC	0.169	0.268	-.351*	0.018	.483**	0.001	-0.057	0.712
Osteomyelitis	0.241	0.111	0.093	0.545	-0.067	0.660	-0.081	0.597
Heart failure	-0.169	0.268	-0.065	0.673	0.047	0.759	0.057	0.712
Avascular necrosis of femoral head	-0.135	0.377	0.065	0.673	-0.047	0.759	-0.057	0.712
Pneumonia	-0.197	0.194	-.322*	0.031	-0.069	0.653	-0.044	0.776
Pigment gallstone jaundice	0.077	0.616	-0.274	0.069	-.318*	0.033	-0.175	0.251
Dehydrated	0.024	0.875	0.093	0.545	-0.067	0.660	-0.081	0.597

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CHAPTER FIVE

DICUSSION, CONCLUSION AND RECOMMENDATIONS

5.1 Discussion

The demographic distribution showed that most of the study participants were female (70%), a trend that is consistent with findings from similar Nigerian studies where women are more likely to attend regular follow-up appointments (Adeyemi *et al.,* 2022). The predominance of Christian respondents in both groups likely reflects the regional population structure rather than any association with the disease. In terms of occupation, a larger proportion of individuals with sickle cell anemia (SCA) were students or self-employed, whereas most participants in the control group were civil servants. This variation may be attributed to the challenges of maintaining steady employment among individuals with chronic illnesses and the physical limitations that often accompany recurrent sickle cell crisis (Nnaji *et al.,* 2021). The observation that most participants with SCA were single also supports earlier research suggesting that chronic illness and concerns about genetic inheritance can influence marital choices (Eke *et al.,* 2020). The high proportion of respondents with tertiary education across both groups indicates good awareness and access to healthcare services, factors that may contribute to better disease understanding and management outcomes.

The analysis of red blood cell (RBC) parameters in this study revealed that sickle cell anemia (SCA) subjects had significantly lower hemoglobin (HGB), packed cell volume (PCV), and red blood cell counts compared to the control group. This aligns with the reports of Olaniyi *et al.* (2021) and Adeyemo *et al.* (2022), who described anemia as a consistent feature of SCA resulting from continuous red blood cell destruction due to sickling and hemolysis. Similarly, Adewoyin *et al.* (2015) and

Akinbami *et al.* (2020) observed markedly reduced hemoglobin and hematocrit levels among Nigerian SCA patients, confirming the chronic nature of anemia in this condition. In contrast, Chikezie *et al.* (2018) found slightly higher hemoglobin values among SCA subjects receiving regular folate supplementation and hydroxyurea therapy, suggesting that management practices can improve hematologic stability.

In contrast to the lower RBC counts, mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) were higher in SCA subjects, indicating reticulocytosis; the release of large, immature, and hemoglobin-rich red cells by a hyperactive bone marrow. This agrees with Nkya *et al.* (2020) and Eke *et al.* (2017), who also attributed elevated MCV and MCH values to bone marrow compensation. Conversely, Okocha *et al.* (2016) reported lower MCV and MCH among SCA patients, likely reflecting coexisting iron or folate deficiency. Furthermore, the significantly elevated red cell distribution width (RDW) in this study aligns with Mohammed *et al.* (2022) and Nwabuko *et al.* (2019**)**, who found that the wide variation in cell size reflects ongoing hemolysis and release of abnormally shaped red cells. The mean corpuscular hemoglobin concentration (MCHC) showed no difference between groups, supporting Ijezie *et al.* (2018), who noted that while the number and size of cells are affected, hemoglobin concentration per cell often remains steady.

When analyzed by body mass index (BMI), these abnormalities persisted across underweight, normal weight, and overweight SCA subjects. This observation mirrors the findings of Olatunji *et al.* (2022) and Okpala *et al.* (2019), suggesting that the hematological disruptions of SCA are independent of nutritional status. Nonetheless, the higher proportion of underweight individuals among SCA subjects supports

Mohammed *et al.* (2022) and Olaniyi *et al.* (2021), highlighting nutritional deficits and increased metabolic demands common among these patients.

White blood cell (WBC) parameters, total WBC counts were significantly higher among SCA subjects compared to controls. This observation is in line with Folarin *et al.* (2023) and Bamidele *et al.* (2021), who associated elevated WBC counts with chronic inflammation and bone marrow stimulation due to recurrent hemolysis. Similarly, Adewoyin *et al.* (2015) and Animasahun *et al.* (2020) reported increased leukocytosis among SCA patients, linking it to infection risk and vaso-occlusive crisis frequency. However, Bello *et al.* (2018) found no significant rise in total WBCs in a cohort of well-managed SCA patients on hydroxyurea therapy, suggesting that medical interventions can modulate these abnormalities. In this study, while differential counts (lymphocytes, granulocytes, and mid cells) showed no significant overall differences, BMI-specific analysis revealed higher lymphocyte and mid-cell percentages but lower granulocyte proportions in SCA subjects. This agrees with Ezeh *et al.* (2019), who also observed lymphocytes predominance in SCA patients, indicating a chronic inflammatory state independent of body weight.

Platelet parameters also showed notable changes. The significantly higher platelet count (PLT) and plateletcrit (PCT) among SCA subjects observed in this study are consistent with Olatunji *et al.* (2022) and Okocha *et al.* (2016), who reported thrombocytosis due to functional asplenia and compensatory marrow overactivity. Similarly, Akinbami *et al.* (2020) noted that persistent platelet elevation contributes to hypercoagulability and vaso-occlusive crises in SCA. Conversely, Nwogoh *et al.* (2018) observed normal platelet counts in hydroxyurea-treated patients, suggesting that therapy can normalize platelet production. In the present study, mean platelet

volume (MPV) and platelet distribution width (PDW) showed no significant differences between groups — a finding comparable to Adewoyin *et al.* (2015), who concluded that while platelet numbers increase, their morphology remains relatively stable.

Iron metabolism indices revealed elevated ferritin levels and reduced total iron-binding capacity (TIBC) in SCA subjects, while serum iron remained normal. This is because the high iron may be effectively trapped in storage within cells due to inflammatory response, preventing it from being released into circulation for use in red blood cell production. This pattern supports Akinyanju *et al.* (2021)**,** Adewole *et al.* (2022)**,** and Saha *et al.* (2020)**,** who attributed similar findings to inflammation-induced iron sequestration rather than actual iron overload. Conversely, Kaur *et al.* (2019) reported elevated serum iron and transferrin saturation among transfused SCA patients, indicating transfusion-related iron accumulation. The uniformity of iron abnormalities across all BMI groups in the present study suggests that inflammatory processes in SCA overshadow nutritional influences, similar to findings by Bello *et al.* (2018) and Eke *et al.* (2017)**.**

BMI distribution patterns revealed that most participants were of normal weight, but underweight individuals were more common among SCA subjects. This aligns with Olaniyi *et al.* (2021) and Mohammed *et al.* (2022), who noted that low BMI in SCA results from increased energy demands, poor appetite, and frequent hospitalizations. Iwalokun *et al.* (2019) similarly observed that despite varying BMI, hematological derangements remain intrinsic to the disease, not merely secondary to nutrition. These consistent findings reinforce that the hematologic and metabolic disruptions in SCA are primarily disease-driven but can be further aggravated by nutritional deficiencies.

Most of the sickle cell anemia (SCA) patients in this study had mild to moderate anemia (about 75.6%). This means that while their hemoglobin levels were low, they were still able to function fairly well in their daily lives. This finding agrees with the report by Ezenwosu *et al.* (2021), who found that many Nigerian adults living with SCA usually maintain hemoglobin levels between **7** and 10 g/dL during steady (non-crisis) periods.

Painful crisis was also very common among participants. Most patients (60%) experienced pain crisis at least once every year, while about 22% had them several times a year. This pattern is similar to what Adisa *et al.* (2023) observed, where vaso-occlusive crisis lead to painful blockages of blood flow due to sickled red blood cells which typically occurred two to four times a year in most adults with SCA.

The major complications reported by participants were vaso-occlusive crisis, acute chest syndrome, and osteomyeliti**s**, which are classic symptoms often seen in SCA patients (Nwogoh *et al.,* 2022). The high rates of acute chest syndrome (50%) and avascular necrosis (48.9%) found in this study highlight the long-term damage caused by repeated inflammation and poor blood flow over time (Machogu *et al.,* 2022). On the other hand, complications such as kidney and heart failure were much less common, which supports previous findings that these problems usually develop in the later stages of the disease (Olayemi *et al.,* 2021).

Pain assessment results further showed that more than 90% of participants experienced pain either “often” or “sometimes**,”** and nearly half said that pain interfered with their daily activities. This clearly shows how painful episodes can

disrupt normal life and reduce overall quality of life for people living with SCA (Nwogoh *et al.,* 2022) as well as economic strength.

5.2 Conclusion

In conclusion, this study demonstrates that SCA is associated with significant alterations in full blood count parameters as well as changes in iron indices metabolism. These abnormalities occur irrespective of BMI status, emphasizing that the hematological impact of SCA is primarily disease-driven rather than weight-related. The findings reinforce that anemia, elevated WBC counts, and thrombocytosis are consistent features of SCA and remain major contributors to disease complications. The findings underscore the need for integrated therapeutic strategies that simultaneously enhance erythropoiesis and modulate inflammatory responses to improve the overall prognosis and quality of life of the affected individuals.

5.3 Recommendations

1. Routine hematological monitoring of SCA patients should include iron indices and inflammation markers to distinguish true iron deficiency from inflammation-related iron sequestration.
2. Nutritional interventions should be incorporated into SCA management programs, focusing on balanced diets and supplementation to address undernutrition and support hematologic health.
3. BMI assessment should be part of regular follow-up, with adequate interpretation among health care workers. Public enlightenment should be offered to sickle cell anemia patients any time of clinic visit, also males should be encouraged to participate in research by clinicians.

4. Further longitudinal and multi-center studies are recommended to explore the long-term relationship between nutritional status, treatment adherence, and hematological responses in SCA patients across different regions.

5.4 Contribution to Knowledge

1. This study provides evidence that the hematological and iron indices abnormalities in SCA are largely independent of BMI.
2. Anthropometric measurement strengthens existing literature by simultaneously analyzing hematologic indices across BMI categories, an aspect not frequently addressed in prior local studies.
3. The study highlights that ferritin elevation in SCA may reflect inflammation rather than iron overload, emphasizing the need for cautious interpretation of iron indices studies in clinical settings.

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APPENDIX I

RESPONDENTS INFORMED CONSENT FORM

Title of Research: Body mass index and Iron indices in Adult Sickle Cell Anemia Patients Visiting LUTH, Idi –Araba, Surulere, Lagos. Lagos State, Nigeria"

Name and affiliation of Researcher: This study is being conducted by Aneke Julian Ngozi, a Postgraduate student of the Department of Medical Laboratory Science, School of Basic Medical Science, College of Medical Sciences, University of Benin, Benin City.

Introduction This is a research project being conducted by Aneke julian Ngozi, a postgraduate student of the University of Benin pursuing an MSc in Medical laboratory Science. I am inviting you to participate in this research if you are currently attending the Sickle cell anemia clinic of the Lagos University Hospital (LUTH), Idi-Araba, Lagos for the management of sickle cell anemia. The aim of this study is to determine the relationship between the BMI and iron indices in adult SCA patients and its impact on severity of the disease. I am therefore requesting your consent to participate in this study.

Purpose of study: BMI is used to know your weight and height. Iron is a mineral present in your red blood cells that caries oxygen; serum iron is used to measure the amount of iron in your blood, total iron binding capacity measures how well the iron moves through your body while serum ferritin stores iron thereby allowing your body to use the iron when need be. Assessment all these will show your health status and better health management.

Procedure of the research: If you consent to participate in this study, an interviewer administer questionnaire that requires you to give information about your clinical condition will be utilizes. It will also involve obtaining 10ml of blood from you to determine your iron indices and full blood count (blood levels)**.**Your weights and heights will also be taken.

Duration: the questionnaires and drawing of blood will take approximately 30 minutes**.**

Potential benefits: Your participation in this study will help understand the association between, body mass index, iron indices and the severity of sickle cell disease.

Risk: You may feel a slight discomfort during blood sample collection.

Voluntarism and right to withdraw without repercussions: Your participation is entirely voluntary. Choosing to participate will not affect you in anyway. Your values will be respected. You may stop participating in this study at any time.

Confidentiality: Be assured that any information provided during the course of this study will be treated with utmost confidentiality and will be used for the research purpose. The name and address of the participants will not be required for this study to maintain anonymity.

Participant's responsibility: You are kindly requested to complete the questionnaires, answer every question honestly.

Statement of person giving informed consent: I have read the description of the research, and understand it. I understand as well that my participation is voluntary. I know enough about the purpose, methods, risk, benefits of the research and I would like to participate. I understand that I can withdraw from the study if need be at anytime. I have received a copy of this consent form for my keep.

Date_____

Signature_____

Name_____

Statement of the person obtaining informed consent: I have fully explained this research to the respondent and given sufficient information, including the risks and benefits, to make an informed decision.

Signature: …… **Date:** ……

Statement of the person giving consent: I have read the description of the research and understood that my participation and withdrawal is voluntary. I am also well informed of the purpose, methods, risks, and benefits of the research study.

Signature: ……

Date: ……..

For more information contact:

Researcher's Contact

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APPENDIX II

Dear Respondents,

You have been selected to participate in this study titled BODY MASS INDEX AND IRON INDICES IN SICKLE CELL ANEMIA PATIENTS VISITING LUTH.IDI-ARABA, LAGOS.

Kindly respond to the questions to the best of your knowledge by ticking.

All information supplied will be kept in strict confidentiality.

Thank you.

QUESTIONNAIRE

Serial No.....

Area of Residence.....

SECTION 1: SOCIO-DEMOGRAPHIC

1. Sex (a) Male (b) Female
2. Age..... years (As at last birthday)
3. Religion: (a) Christianity (b) Islam (c) Traditional (d) Others (specify).....
4. Occupation: (a) Civil Servant (b) Self Employed (c) House Wife (d) Retiree (e) Others specify.....
5. Education: (a) No Formal Education (b) Primary (c) Secondary (d) Tertiary (e) Others specify.....
6. Ethnicity (a) Igbo (b) Yoruba (c) Hausa (d) Others specify.....
7. Current Marital Status(a) Married (b) Single (c) Separated (d) Divorced (e) Widowed
8. How long have you been diagnosed of sickle cell anemia.....
9. In general, would you say your health is (a) Excellent (b) Good (c) Fair (d) Poor
10. Have you ever been transfused (a) Yes (b) No
11. How many times of transfusions in life time.....
12. How many transfusions in the last one year.....
13. When was the last transfusion.....

SECTION 2: Disease Severity Scoring System for Sickle Cell Anemia Patients

14. How often do you have painful crisis (a) Weekly (b) Monthly (c) Bimonthly (d) Quarterly (e) Yearly

15. What type of crisis do you have and how many times per year (1-10 times)

(a) VOC Yes ☐ No ☐

(b) Sequestration Yes ☐ No ☐

(c) Acute chest syndrome Yes ☐ No ☐

(d) Osteomyelitis Yes ☐ No ☐

(e) Renal failure Yes ☐ No ☐

(f) Heart failure Yes ☐ No ☐

(g) Avascular necrosis of femoral head Yes ☐ No ☐

(h) Pneumonia Yes ☐ No ☐

(i) Pigment gallstone jaundice Yes ☐ No ☐

(j) Dehydrated Yes ☐ No ☐

(k) Anemia/ Hb ≥ 10g/ dl

Hb ≥ 8 < 10g/dl

Hb ≥ 6 < 8g/dl

Hb ≥ 4 < 6g/dl

Hb ≤ 4g/dl

SECTION 3: Pain Assessment Report in Sickle Cell Anemia Patients

	Always	Often	Sometimes	Rarely	Never
16. In the past 7 days; (a) How often did you have pain so bad that you	☐	☐	☐	☐	☐

could not do anything for a whole day?

(b) How often were you totally pain free? ☐ ☐ ☐ ☐ ☐

	Always	**Often**	**Sometimes**	**Rarely**	**Never**
(c) How often did you have a sudden attack of severe pain?	☐	☐	☐	☐	☐
(d) How often did you have pain so bad that you could not get out of bed?	☐	☐	☐	☐	☐
(e) How often did you have very severe pain?	☐	☐	☐	☐	☐
(f) How often did you cancel plans because of pain?	☐	☐	☐	☐	☐
(g) How often did you have pain so bad that you had to stop what you were doing?	☐	☐	☐	☐	☐

(h) How often did you have pain so bad that it was difficult to finish what you were doing? ☐ ☐ ☐ ☐ ☐

(i) How often were you terrified that you might have a pain attack (crisis)? ☐ ☐ ☐ ☐ ☐

(j) How bad was the pain you usually have? ☐ ☐ ☐ ☐ ☐

(k) How bad was the pain in your joints such as hips or shoulder? ☐ ☐ ☐ ☐ ☐

(l) How many days did pain prevent you from doing anything? ☐ ☐ ☐ ☐ ☐