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Human Immunodeficiency Virus Co-Infection among Sickle Cell-Exposed Children in Kisumu County, Western Kenya

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Abstract:
Globally, the high prevalence of infectious diseases, particularly HIV, and the possible interaction with sickle cell disease, has been of great public health concern. Kenya experiences a significant burden of HIV. Kisumu County in particular, exhibits unique epidemiological burden characterized by unprecedented high mortality 50%-90% among children with sickle cell disease,. This is coupled with high prevalence 17.5% of HIV/AIDS among its population, and high malaria endemicity with prevalence of 19%. The overarching aim of this study was to assess the HIV co-infection among sickle cell-exposed children in Kisumu County. The study adopted a retrospective systematic review. Study population comprised of the sickle cell-exposed children in Kisumu County with a sample size of 173. The study used both purposive and simple random sampling techniques. Majority 99.2% of the sample size participated. The prevalence of HIV among sickle cell-exposed children was 4.65% in Kisumu County. Majority 76.3% sickle cell-exposed children had hemoglobinopathies for sickle cell disease. Age was not statistically significant factor p-value= 0.414 complicating co-infection. A paltry one quarter 49 (28.48%) had attained fifth anniversary with close to a third 47 (27.33%) being only 7 months to one and half years old. Viral load was statistically significant p-value=0.046 in β-thalassemia co-infection. The study recommends enhancement of HIV screening and prevention efforts in this vulnerable population

1. Introduction

1.1 Background of the Study

Human Immunodeficiency Virus (HIV) and Sickle Cell Disease (SCD) represent a significant global health burden. Approximately 5% of the global population carries the sickle cell trait, with a higher concentration observed in malaria-endemic regions (Piel *et al.*, 2013). Concurrently, HIV remains a pervasive global health crisis. World Health Organization, 2020, estimated that 38 million people worldwide, were living with HIV, with roughly 1.5 million new infections and 680,000 acquired immune deficiency syndrome-related deaths in that year.

Sub-Saharan Africa, a region characterized by both heightened burdens of SCD and HIV, faces unique health challenges. It bears a disproportionate burden, accounting for about 67% of global HIV infections (UNAIDS, 2020). The prevalence of SCD is notably high, with approximately 2% of births affected by the disease, contributing to substantial morbidity and mortality (Piel *et al.*, 2013). Furthermore, Sub-Saharan Africa harbors about 20.6 million people living with HIV, underscoring the urgent need for comprehensive and region-specific healthcare interventions (UNAIDS, 2020). The highest prevalence of HIV among children with sickle cell trait has been reported to be 11.5% in Sub-Saharan Africa with the infections regarded as endemic and overlapping (Owusu *et al.*, 2014).

In Kenya, the prevalence of SCD is characterized by high level genetic diversity, with a national estimate of the sickle cell trait at 13.3% (Makani *et al.*, 2011). On the other hand, Kenya, like other sub-Saharan African countries, continues to grapple with the HIV epidemic, with an estimated 1.5 million (4.9%) people living with HIV (UNAIDS, 2020).

Kisumu County exemplifies the local dynamics of SCD and HIV prevalence. The prevalence of sickle cell trait and SCD has been reported to be 17.5% and 1.2% respectively among neonate in Kisumu County (Were *et al.* 2018).

These statistics underscore the genetic complexity and significant impact of SCD within the local population. Concurrently, Kisumu County is identified as a high-burden HIV region, recording a prevalence rate of 17.3% in 2018 (KAIS, 2018).

The compounded health challenges faced by sickle cell-exposed children in Kisumu County demand meticulous investigation. According to Odera *et al.*, (2014) there are management challenges faced by children with both conditions in resource-limited settings. Kumari *et al.*, (2021) on the other hand indicated restriction of HIV-1 infection in individuals with the sickle cell trait.

Sickle cell disease (SCD) is a genetic blood disorder characterized by the presence of abnormal hemoglobin, known as hemoglobin S (HbS). This inherited condition primarily affects red blood cells, causing them to assume a crescent or sickle shape instead of the normal, round shape. This altered structure can lead to various complications due to the increased fragility and reduced flexibility of the sickle-shaped cells.

One of the primary effects of sickle cell disease is the occlusion of small blood vessels by these misshapen red blood cells, resulting in vaso-occlusive crises. These painful episodes can cause damage to organs, tissues, and bones, leading to chronic pain, organ dysfunction, and a decreased quality of life for individuals with SCD. Additionally, the altered red blood cells have a shorter lifespan, leading to chronic anemia, fatigue, and an increased risk of infections (CDC, 2023).

The relationship between sickle cell disease and HIV/AIDS is complex, as both conditions can independently impact the immune system and overall health. Individuals with SCD may experience immune system dysfunction, making them potentially more susceptible to infections. Acquired immunodeficiency syndrome, caused by HIV, specifically targets and weakens the immune system, compromising the body's ability to fight off infections (Kelly *et al.*, 2020).

When individuals with sickle cell disease are co-infected with HIV, the interaction between these conditions can pose additional challenges. Immune compromise associated with HIV may exacerbate the health complications already present in individuals with SCD. Infections may become more severe, and the frequency and severity of vaso-occlusive crises may increase in co-infected individuals (Egesa et al., 2022).

The understanding of the relationship between sickle cell disease and HIV/AIDS is an evolving field, and more research is needed to delineate the specific interactions and impacts of co-infection. Addressing the healthcare needs of individuals with both conditions requires a comprehensive and integrated approach, considering the unique challenges posed by each disorder and their potential synergistic effects. Research findings on this topic may contribute to the development of targeted interventions and improved healthcare strategies for individuals living with both sickle cell disease and HIV/AIDS. The proposed research seeks to contribute essential data on the seroprevalence of HIV and characterization of hemoglobin types and the intricate dynamics of correlation between HIV viral load and hemoglobin types in sickle cell conditions. Understanding the complex interplay of SCD and HIV is vital for the formulation of evidence-based interventions and policies tailored to the distinctive health challenges confronted by sickle cell-exposed children in Kisumu County.

1.2 Statement of the Problem

Kisumu County grapples with a pronounced epidemiological conundrum characterized by the co-occurrence of SCD and HIV among its pediatric populace. The amalgamated health burden necessitates precise, evidence-driven interventions to address the unique clinical exigencies of this vulnerable demographic.

Empirical data gleaned from the study by Were *et al.* (2018) underscores a notable prevalence of sickle cell trait at 17.5% and sickle cell disease at 1.2% among neonates in Kisumu County, thereby spotlighting the salient impact of SCD in the region. The concurrent presence of HIV is an additional layer of complexity to the Kisumu County's health landscape, characterized by a documented HIV prevalence rate of 17.3% (KAIS,2018).

This elevated HIV prevalence underscores the need for scientific inquiry into the dynamics surrounding HIV infection within the context of sickle cell-exposed children. Children exposed to sickle cell conditions are vulnerable to a myriad of health issues, and co-infection with HIV further complicates their health outcomes. The adverse effects of HIV infection among sickle cell-exposed children are not well-understood within the local context, posing challenges to effective healthcare interventions. Despite the growing recognition of the coexistence of HIV and sickle cell disease, and the effects, there is scarcity of data on the seroprevalence of HIV and the specific hemoglobin types among sickle cell-exposed children in Kisumu County. This research seeks to address this critical gap in knowledge.

Understanding these dynamics is crucial for tailoring interventions and optimizing healthcare delivery to improve the well-being of children affected by both HIV and sickle cell conditions. This research therefore aims to bridge this gap by providing understanding of the intersection between HIV and sickle cell disease in a specific geographic and demographic context.

1.3 Justification of the Study

The paramount significance of this study in Kisumu County emanates from its distinctive role as both a hot spot for HIV transmission and a region characterized by a notable prevalence of sickle cell disease and accentuates the urgency of comprehending the specific dynamics associated with co-infection. This research addresses a critical lacuna and unravels the intricacies of healthcare needs concerning the nuanced co-infection dynamics within this specific geographic context. Additionally, the study hopes to offer imperative insights for crafting interventions that meticulously consider the coalescence of these health challenges, offering valuable insights into the global understanding of co-infection dynamics between HIV and sickle cell disease.

The findings may well have implications for analogous regions worldwide, thereby fostering a more comprehensive approach to addressing the intricate healthcare needs of individuals grappling with both conditions. In essence, this scholarly pursuit stands as a crucial stride towards bridging the knowledge gap, advocating for tailored interventions, and acknowledging the coexistence of HIV and sickle cell disease in Kisumu County and beyond.

1.4 Research Questions

1. What are the seroprevalence of HIV among sickle cell-exposed children in Kisumu County, Western Kenya?

3. What are the influence of HIV co-infection among sickle cell-exposed children in Kisumu County, Western Kenya

1.5 Objectives

This is composed of broad objective and specific objectives.

1.5.1 Broad Objective

To assess the HIV co-infection among sickle cell-exposed children in Kisumu County, Western Kenya.

1.5.2 Specific Objectives

1. To establish the seroprevalence of HIV among sickle cell-exposed children in Kisumu County, Western Kenya

2. To determine the influence of HIV co-infection among sickle cell-exposed children in Kisumu County, Western Kenya

1.6 Significance of the Study

This study holds crucial public health significance in understanding the dynamics of HIV and SCD co-infection. The study finding will offer evidence-based insights for healthcare policies, optimization of healthcare delivery through effective planning and development of intervention strategies.

Literature Review

2.1 Overview of HIV and Sickle Cell-Condition

Human Immunodeficiency Virus and sickle cell conditions, encompassing both sickle cell trait (SCT) and sickle cell disease (SCD), present intricate health challenges (David *et al.*, 2018). A thorough exploration of the etiopathogenesis of each condition, the dynamics of co-infection, the effects of their co-occurrence, research findings on this intersection, existing management guidelines, and appropriate interventions to mitigate adverse effects are essential for a comprehensive understanding of these complex health issues.

2.1.1 Etiopathogenesis of HIV and Sickle Cell Condition

Human Immunodeficiency Virus, a retrovirus, targets the immune system by infecting CD4 T cells. The virus enters host cells, replicates, and progressively weakens the immune system. Transmission primarily occurs through unprotected sexual intercourse, sharing of contaminated needles, or from an infected mother to her child during childbirth or breastfeeding.

Sickle cell conditions on the other hand result from a genetic mutation in the HBB gene. Sickle Cell Trait involves carrying one abnormal hemoglobin gene, while SCD involves inheriting two, leading to the production of abnormal hemoglobin S (HbS). This mutation causes red blood cells to assume a sickle shape, resulting in complications such as vaso-occlusive crises and chronic anemia (Kelly *et. al.*, 2019).

Co-infection with both HIV and sickle cell conditions introduces additional complexities. Individuals with SCD, even in the trait form, may experience increased susceptibility to infections, compounded by the immunocompromised state induced by HIV. Co-infection may lead to more severe infections and heightened risks of vaso-occlusive crises, particularly in individuals with SCD.

The co-occurrence of HIV and sickle cell conditions can lead to a range of complications. Individuals with SCT or SCD may have an increased vulnerability to infections, and the immunocompromised state induced by HIV can exacerbate these effects. Co-infected individuals may experience more severe infections and an elevated risk of vaso-occlusive crises, impacting overall health outcomes (Owusu, *et al.*, 2014).
However, the understanding of the specific interactions, mechanisms, and long-term effects of co-infection remains an evolving field, necessitating further research to delineate the nuances of this intersection.

2.1.2 Management of Co-infection of HIV and Sickle Cell-condition

Comprehensive management guidelines for individuals co-infected with HIV and sickle cell conditions are not universally established. Management typically involves addressing each condition separately, considering antiretroviral therapy (ART) for HIV and disease-modifying therapies, such as hydroxyurea, for SCD. Regular medical monitoring is crucial to assess disease progression and manage complications. Vaccination against infections is also recommended to prevent additional health challenges.

Strict adherence to antiretroviral therapy (ART) is essential for individuals with HIV. For sickle cell conditions, disease-modifying therapies like hydroxyurea, which has been shown to reduce vaso-occlusive crises, should be considered. Managing each condition optimally is fundamental to reducing adverse effects. Co-infected individuals should undergo regular medical monitoring to assess the progression of both HIV and sickle cell conditions (Were *et. al.*, 2014). This includes routine blood tests, viral load measurements, and monitoring of hemoglobin levels to identify and address any complications promptly. Adequate pain management strategies are crucial for individuals with co-infection, especially considering the increased frequency and severity of vaso-occlusive crises. Tailored pain management plans, including medications and supportive care, can significantly improve the quality of life.

Vaccination against common infections, including influenza and pneumococcus, is essential for individuals with compromised immune systems. Immunizations can reduce the risk of additional complications in co-infected individuals. Comprehensive psychosocial support is essential for individuals living with co-infection. Counseling services can address mental health challenges, and support groups provide a platform

for sharing experiences and coping strategies. Genetic counseling is crucial for individuals with co-infection, especially those with SCT, to understand the hereditary aspects and make informed decisions about family planning. This involves discussing the risk of passing SCT or SCD to offspring and exploring available options.

2.2 Seroprevalence of HIV among sickle cell-exposed children

The intersection of Human Immunodeficiency Virus (HIV) and Sickle Cell Disease (SCD) among children presents a complex landscape requiring comprehensive exploration. This literature review delves into the seroprevalence of HIV/AIDS among children with sickle cell trait and sickle cell disease, elucidating the burden, effects, correlation, other findings, challenges, and interventions pertinent to this intricate interplay. The burden of HIV/AIDS among children with sickle cell conditions is a multifaceted challenge. According to a study by Kumari *et al.* (2021), the prevalence of sickle cell trait among HIV-positive children was reported at 18.8%, Lagos, Nigeria. This high prevalence underscores the substantial burden faced by children co-affected by these conditions. The coexistence of HIV and sickle cell conditions accentuates the complexity of healthcare management and necessitates targeted interventions to address the unique challenges encountered by this population.

Understanding the burden involves not only assessing prevalence rates but also considering the socioeconomic and healthcare disparities that may exacerbate the challenges faced by children with both HIV/AIDS and sickle cell conditions. The burden extends beyond the individual health of affected children to encompass broader implications for healthcare systems and resource allocation, particularly in regions with high prevalence rates.

The effects of the simultaneous presence of HIV/AIDS and sickle cell conditions are intricate and extend across various dimensions. Kelly et al. (2019) conducted research focusing on the molecular intricacies of this interaction. Their study revealed potential factors influencing susceptibility to HIV infection in individuals with sickle cell disease. At the molecular level, the effects of the interplay between HIV and sickle cell disease manifest in intricate ways, impacting cellular processes and immune responses.

Beyond the molecular realm, the effects of these coexisting conditions may contribute to a compounding of health challenges. Children with sickle cell disease often experience chronic pain, anemia, and increased vulnerability to infections. The immunocompromised state induced by HIV further exacerbates these challenges, leading to a complex clinical picture that demands specialized care and management. Correlating the prevalence of sickle cell conditions, particularly sickle cell trait, with HIV/AIDS among children provides valuable insights. David et al. (2018) reported a prevalence rate of 18.8% for sickle cell trait among HIV-positive children, indicating a notable correlation.

This finding underscores the importance of simultaneous consideration of both conditions in clinical assessments and epidemiological studies. Understanding the correlation between sickle cell conditions and HIV/AIDS is essential for tailoring healthcare interventions. The shared demographic factors, such as geographic location or socioeconomic status, may contribute to the observed correlation. Moreover, exploring potential genetic correlations could unveil shared pathways or vulnerabilities, influencing susceptibility to both conditions.

According to Waweru *et al.*, (1988) historical study, the only one in Kenya on seropositivity of HIV in sickle cell anaemia, though limited in specifics, signifies an early acknowledgment of this intersection during the initial stages of the HIV epidemic. The study laid the groundwork for subsequent research endeavors and serves as a historical anchor in understanding the evolving dynamics of these conditions over time. Kumari *et al.*(2021) work on sickle cell trait individuals provides additional genetic insights. The study explored into the restriction of HIV-1 infection in sickle cell trait individual. This genetic perspective suggests varying susceptibility within the broader sickle cell-exposed cohort.

The coexistence of HIV/AIDS and sickle cell conditions introduces a unique set of challenges in the care and management of affected children. The management challenges encompass not only medical aspects but also psychosocial and educational dimensions. Children with both HIV/AIDS and sickle cell conditions may

require specialized care teams, including hematologists, infectious disease specialists, and mental health professionals. The intricate nature of these challenges demands a multidisciplinary and holistic approach to healthcare delivery (Owusu *et al.,* 2014).

A. It is crucial to develop effective interventions and tailored healthcare strategies for addressing the dual burden of HIV/AIDS and sickle cell conditions among children. Interventions including routine screening, comprehensive healthcare programs, and genetic counseling to empower families with knowledge about potential risks and protective measures. Educational programs for both healthcare providers and affected families are crucial in fostering understanding and adherence to treatment regimens. Research into novel therapeutic approaches, including gene therapies or targeted antiretroviral strategies offer promising avenues for intervention.

2.3 Influence of HIV co-infection among sickle cell-exposed children

The intricate interplay between HIV viral load and hemoglobin types in children exposed to sickle cell conditions represents a complex yet crucial aspect of biomedical research.

To understand the association between HIV viral load and hemoglobin types in sickle cell-exposed children, it is paramount to acknowledge the heightened vulnerability of this demographic. According to Owusu *et al.* (2014), the dual burden necessitates a deeper comprehension. This will ensure effective clinical interventions. This highlights the criticality of investigating the intricate relationship between viral load and the diverse hemoglobin types in this cohort.

Pioneering studies by Steinberg *et al.* (2001) and Ballas *et al*. (2012) meticulously delineate distinctions between sickle cell disease (SCD) and sickle cell trait (SCT). This accentuates the diversity of hemoglobin compositions within these categories. The presence of hemoglobin S (HbS) in SCD and HbA in SCT forms the molecular backdrop for understanding how these variations intricately influence the dynamics of HIV viral load.

Complementing the molecular understanding, Kelly *et al.* (2019) enrich the literature with insights into the complexities governing the interaction between sickle cell disease and susceptibility to HIV infection. This molecular perspective augments our understanding of how hemoglobin types, particularly the polymerization of abnormal hemoglobin such as HbS in sickle cell disease modulates cellular processes and immune responses, potentially influencing HIV viral replication dynamics. This molecular lens illuminates the intricate terrain upon which the association between hemoglobin types and HIV viral load unfolds.

David *et al.* (2018) provide insights into the prevalence and impact of sickle cell trait on the clinical and laboratory parameters of HIV-infected children in Lagos, Nigeria. The study underscores the necessity of considering sickle cell trait, specifically hemoglobin AS (HbAS), in the context of HIV.

The reported prevalence of sickle cell trait among HIV-positive children, standing at 18.8%, implies a substantial representation of this hemoglobin type within the population. This research spotlights the potential implications of specific hemoglobin types, even in the trait state, on the clinical trajectory of HIV and accentuates the need for nuanced investigation into the nuances of this association. Exploring the influence of sickle cell disease on susceptibility to HIV infection, Kelly *et al.* (2015) scrutinize the role of hemoglobin types, notably HbS in sickle cell disease.

The findings suggest that individuals with sickle cell disease exhibit altered susceptibility to HIV due to the unique molecular and cellular environment shaped by the presence of abnormal hemoglobin. This emphasizes the imperative to consider not only the presence of HIV but also the potential impact of specific hemoglobin types on susceptibility, progression, and viral dynamics.

According to Kumari *et al.,* (2021) the restriction of HIV-1 infection in sickle cell trait individuals introduces a genetic dimension to the association between hemoglobin types and HIV viral load. The study explored the genetic resilience conferred by the sickle cell trait, unveiling potential mechanisms that restrict HIV-1 infection. The genetic factors associated with specific hemoglobin types exert a protective effect, influencing the dynamics of HIV viral load among individuals with sickle cell exposure. This genetic perspective adds layers to our understanding, suggesting a complex genetic terrain that necessitates meticulous exploration.

The correlation between hemoglobin types and disease severity extends beyond the susceptibility to HIV to encompass the overall clinical impact of sickle cell conditions. Charache *et al.,* (1995) delve into the association between different hemoglobin types and the frequency of painful crises, a hallmark complication of sickle cell disease. Their findings imply that individuals with hemoglobin SC (HbSC) experience less frequent and less severe painful crises compared to those with HbSS. Comprehending this correlation is paramount not only in understanding the clinical manifestations of sickle cell conditions but also in deciphering their potential influence on the course of HIV infection and the dynamics of viral load.

The therapeutic implications of understanding the association between HIV viral load and hemoglobin types are illuminated by Tshilolo *et al,* (2019).

This study focused on the administration of hydroxyurea, an inducer of fetal hemoglobin (HbF) production, to individuals with sickle cell anemia. This therapeutic intervention, tailored based on specific hemoglobin types, showcased the potential to ameliorate clinical symptoms and improve.

2.5 Conceptual Framework

The conceptual framework was anchored on Health Belief Model (HBM). The model focuses on individual characteristics, risk factors, and the outcome from the implemented intervention strategies. The independent variables included statuses of HIV, and Sc, and also the HIV/SC co-infection. Intervening variables was the HIV viral load. The dependent variables included the prevalence of HIV and SC infections.

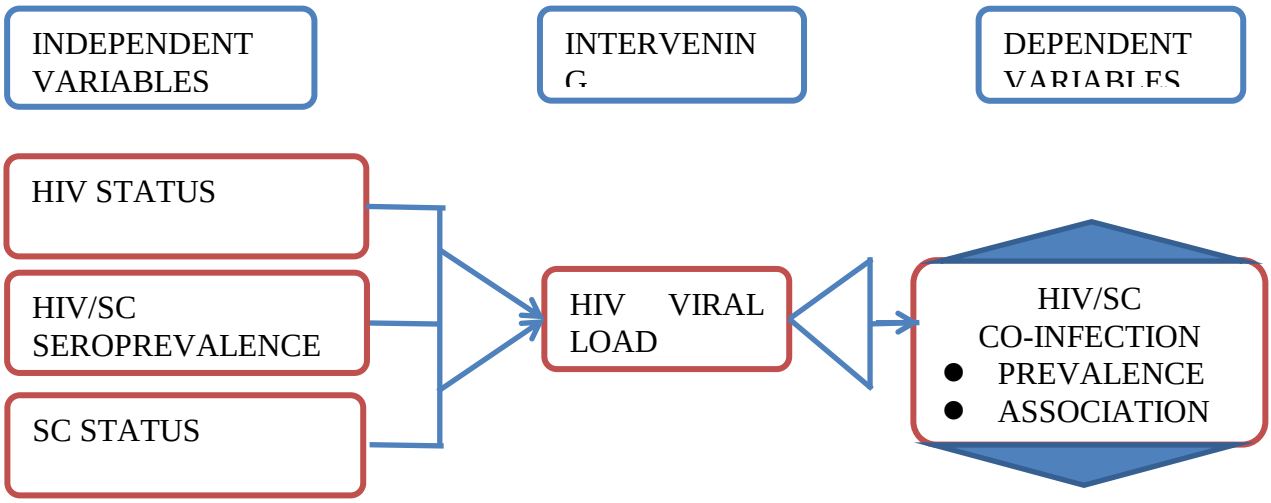

Fig 1: Conceptual Framework

Methodology

3.1 Study Area

This research was carried out at Obama children clinic at Jaramogi Oginga Odinga Teaching and Referral Hospital in Kisumu County. The Obama children clinic was chosen because it is the only established sickle cell clinic in Kisumu County and the only existing one in Western Kenya region. Kisumu county is one of the 47 counties in the Republic of Kenya. The county is located in Western Kenya with its headquarters in Kisumu City, the third largest city in Kenya. The county has six bordering counties: Siaya County to the West, Vihiga County to the North, Nandi County to the North East, and Kericho County to the East. On the southern border is Nyamira County, and Homa Bay County is to the South West. It has a shoreline on Lake Victoria, occupying northern, western and a part of the southern shores of the Winam Gulf. Kisumu County has a population of 1,155,574 (2019 National Census). The land area of Kisumu County totals 2085.9 km^2. Administratively, the County is divided into seven sub counties and 35 wards.

3.2 Study Design

This research adopted a retrospective systematic review.

3.3 Study Population
The study population comprised of the sickle cell-exposed children who had attended Obama children clinic at JOOTRH within the last one year.

3.4 Inclusion and Exclusion Criteria
The criteria included;

3.4.1 Inclusion Criteria
The criteria included:
1) Children who were either sickle cell disease or sickle cell trait
2) Children who were below 18 years old
3) Children who were residents of Kisumu County
4) Children who attended Obama clinic at JOOTRH within the last one year
5) Children who had been diagnosed or confirmed by JOOTRH as having either sickle cell disease or sickle cell trait within the last one year

3.4.2 Exclusion Criteria
The following were excluded:
1) Children who had neither sickle cell disease nor sickle cell trait
2) Those who were above 18 years old
3) Children who were not residents of Kisumu County
4) Children who had not attended Obama clinic at JOOTRH within the last one year
5) Children who had not been diagnosed or confirmed by JOOTRH as having either sickle cell disease or sickle cell trait within the last one year

3.5 Sampling Techniques
In this retrospective study, the registers for children who had attended at Obama clinic during the last one year (2023/2024) were selected purposively. Simple random sampling was used to pick up all the 173 children below 18 years who were having either sickle cell disease or sickle cell trait. This was done from the latest dates backwards.

3.6 Sample Size Determination
This study's sample size was determined using a formula adopted from Cochran's formula (1977). This made it possible to determine the optimal sample size for a defined degree of accuracy, degree of confidence, and estimated percentage of the characteristic present in the population. Both big and small populations are taken into consideration. The formula is:

$$n = \frac{(Z\alpha/2)^2(PQ)}{d^2}$$

Where:

n = the sample size (No. of children whose records were selected)
d = 0.05 (the sampling margin error (5%) that was accepted in this study
Z α/2=1.96 (Z score corresponding to a 95% confidence interval)
P=0.115 (estimated proportion of children with sickle cell and HIV co-infection in Sub-Saharan Africa).
Q = 0.885 (1-P)

Substitution.

$$n = \frac{(1.96)^2(0.115x0.885)}{0.05^2}$$

$$=156.39$$

According to (UNFPA, 2014), 10% of n was added for incomplete data. 10% of 156.39 equals 15.639. The adjusted sample size, taking into account the incomplete data was 172.03 HIV-exposed children. The rounding off of 172.03 to 173 was done because there is no half human being. The adjusted sample size (n) in this study was now =173.

3.7 Data Collection Tools

The data from the records was captured using structured questionnaires designed to capture socio-demographic characteristics: age and gender, and health characteristics: HIV status, percentage hemoglobin types, sickle cell status, HIV-viral load, and age at viral load test. Other tools for data collection included: Registers, pens, note books, printers, laptops, and electronic records.

3.8 Data Collection Procedure and Processing

The electronic register for children who had attended Obama clinic between the year 2023 and 2024 was selected. A line-list of 173 children having either sickle cell disease or sickle cell trait was generated. This was done through random selection of all children who had either sickle cell or sickle cell trait identifier. The selection was done beginning from the month of March, 2024 backwards until the sample size of 173 was achieved. In the line-list, the last unique number represented a child who was diagnosed in the month of January, 2023. The unique numbers were selected then used to generate electronic reports on hemoglobin types, hemoglobin percentages, HIV status, and HIV viral load. HB Electrophoresis (HBE) was done using HPLC-D10 automated machine and used to determine the types and percentage of hemoglobin types. The process HBE involved aliquoting 3ml of whole blood collected in purple vacutainer. The HPLC-D10 sucked and processed the sampled specimen and generated result in form of hemoglobin types and their percentages. The PCR machine generated the HIV RNA copies/ ml of whole blood for viral load.

3.9 Pilot Study, Reliability, and Validity

A pilot study was conducted with 10 samples from the year 2022 from the electronic records of Obama Clinic to assess the feasibility and clarity of data collection tools. Feedback from the pilot study was used to refine the questionnaires and ensure they were contextually appropriate. Face and content validity of the data collection tools was assessed through peer review. Additionally, pre-testing and piloting of the tools contributed in ensuring the validity.

Inter-rater reliability for questionnaire administration was assessed through training and periodic checks. Operating procedures was standardized and conducted to ensure the reliability of biological measurements.

3.10 Data Management and Analysis

The report was collated into dataset, and coded. Data was entered into a secure electronic database: Google Cloud SQL. The analysis was done using SPSS, and STATA version 13. Descriptive statistics was used to summarize demographic characteristics, seroprevalence rates, the hemoglobin types. Inferential statistics, P-value, odds ratio, and regression analysis, were employed to explore significance and degree of associations between sickle cell disease, and the seroprevalence of HIV.

B.

3.11 Ethical Considerations

Ethical approval was obtained from the JOOTRH-ISERC prior to data collection. License was sought from NACOSTI, approval of the JOOTRH CEO, and County Government Kisumu County was also obtained. Consent was neither verbal nor written. It was not applicable as the data was analyzed anonymously from medical record registers. Medical laboratory officers handling the registers were verbally informed of the essence of the research. The address for the PI: P.O Box 2268-40100 Kisumu. The contact for IRB is JOOTRH-ISERC P.O Box 849-40100 Kisumu..

Results

4.1 S**ocio-demographic characteristics of the children**

Table 1 below comprises information on the socio-demographic distribution of children across various age cohorts and their corresponding gender proportions. There were a total of 172 sickle cell-exposed children participants in this study. The average age-cohort was 7 months to 19 months. The median age-cohort was 33 months to 45 months.

Majority of children fell within the age range of 7 to 19 months, constituting 27.33% (n = 47) of the total sample size of 172. The second largest age group consisted of children aged 33 to 45 months, representing 17.44% (n = 30) of the total. The smallest age group were children aged 85 to 97 months, comprising only 0.58% (n = 1). Other age categories: 20 to 32 months were 11.63% (n = 20), 46 to 58 months 10.47%, (n = 18), and 59 to 71 months 6.4%, (n = 11). Conversely, the age bracket encompassing children older than 123 months constituted 9.88% (n = 17).

Table 1 : *Age and Gender distribution of the children*

Variables	**N (%)**	**Female n (%)**	**Male n (%)**
Age			
<6 months	8(4.65)	5(71.43)	2(28.57)
7 to 19 months	47(27.33)	14(35.9)	25(64.1)
20 to 32 months	20(11.63)	8(44.44)	10(55.56)
33 to 45 months	30(17.44)	12(44.44)	15(55.56)
46 to 58 months	18(10.47)	8(47.06)	9(52.94)
59 to 71 months	11(6.4)	4(50)	4(50)
72 to 84 months	7(4.07)	1(20)	4(80)
85 to 97 months	1(0.58)	1(100)	0(0)
98 to 110 months	8(4.65)	2(33.33)	4(66.67)
111 to 123 months	5(2.91)	2(40)	3(60)
> 123 months	17(9.88)	11(64.71)	6(35.29)
Overall	172 (100.00)	68(45.38)	82(54.67)
Children whose gender were not indicated =22 (12.79%)			

In terms of gender distribution, out of the total sample of 172 sickle cell-exposed children, 68 (45.38%) were females, and 82 (54.67%) were males. However, 22 sickle cell-exposed children (12.79%) had unspecified gender information. The percentage of female children varied across age groups. In the age-bracket under 6 months, female children accounted for 71.43% (n = 5). Male children dominated most of the age groups. Among children aged 72 to 84 months, 80% (n = 4) were males, representing the highest male percentage within any age group. Additionally, in the age group of 7 to 19 months, males constituted the largest proportion, with 64.1% (n = 25).

Fig 2 below shows that more than three-quarters (76.16%, n=131) of sickle ell-exposed children suffer sickle cell disease. Slightly more than a fifth (23.84%, n=41) had sickle cell trait status.

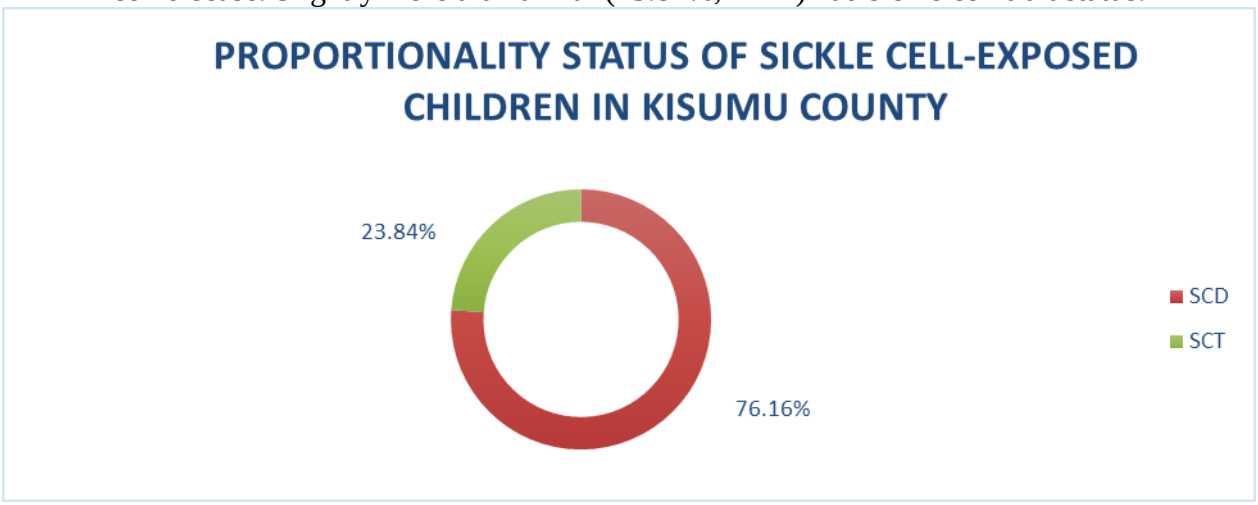

Fig 2: Doughnut showing the proportionality status of sickle cell-exposed children

The findings from table 2 below shows that the age cohort between 7 months to 19 months had the highest number of children affected either with sickle cell disease (29.01%, n=38), or trait (21.95%, n=9).

Table 2: *Sickle Cell Status Based on Age*

VARIABLES	**Age in Months**	<6	7-19	20-32	33-45	46-58	59-71	72-84	85-97	98-110	111-123	> 123
	N (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
SCD	131 (76.16)	5 (3.82)	38 (29.01)	15 (11.45)	23 (17.56)	13 (9.92)	9 (6.87)	5 (3.82)	1 (0.76)	7 (5.34)	4 (3.05)	11 (8.4)
SCT	41 (23.84)	3 (7.32)	9 (21.95)	5 (12.22)	7 (17.07)	5 (12.2)	2 (4.88)	2 (4.88)	0 (0)	1 (2.44)	1 (2.44)	6 (14.63)

Sickle cell-exposure among children below 6 months were a paltry (3.82%, n=5). The least was the age cohort between 85 months to 97 months with less than one percent (0.76%, n=1) sickle cell disease cases. There was no child with sickle cell trait in the 85 months to 97 months cohort. More children with sickle cell trait (14.63%, n=6) were in age cohort >123 months than children with sickle cell disease (8.4%, n=11).

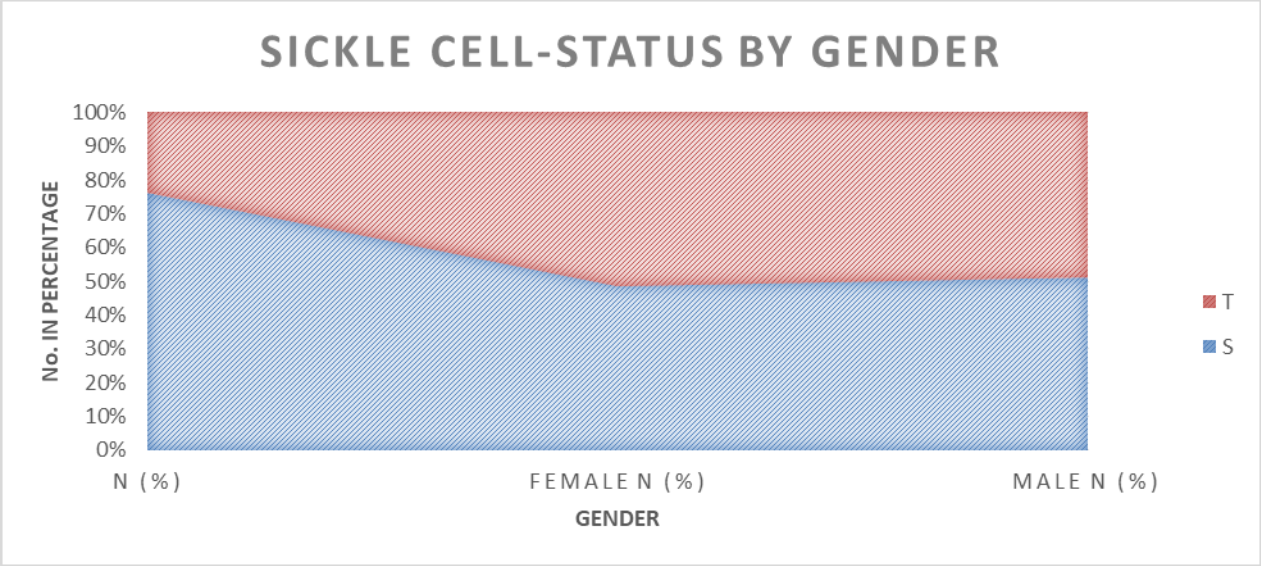

Fig 3: Gender-Based status of sickle cell-exposed children

Figure 3 above is an area chart showing the sickle cell-exposure status based on gender. The female children who had sickle cell disease were (34%, n=51) less than those female children who were having sickle cell trait (11.3%, n=17).More male children had sickle cell disease (42%, n=63).

The male children having sickle cell trait were however more (12.7%,n=19) than the female counterpart. Overally, the male children were more exposed to sickle cell disease (54.7%, n=82) than the male children. This was the same as the exposure to sickle cell trait. The female children were less exposed (11.3%, n=17) as the male children (12.7%, n=19).

4.2 Seroprevalence of HIV among sickle cell-exposed children

This study sought to determine the rate of HIV-Sickle Cell co-infection. Figure 4 below is a pie-chart that shows the HIV seroprevalence among children who are bearing the burden of sickle cell in Kisumu County. The findings are that close 5 children out of 100 sickle cell-exposed children (4.65%) suffer additional HIV infection in Kisumu County.

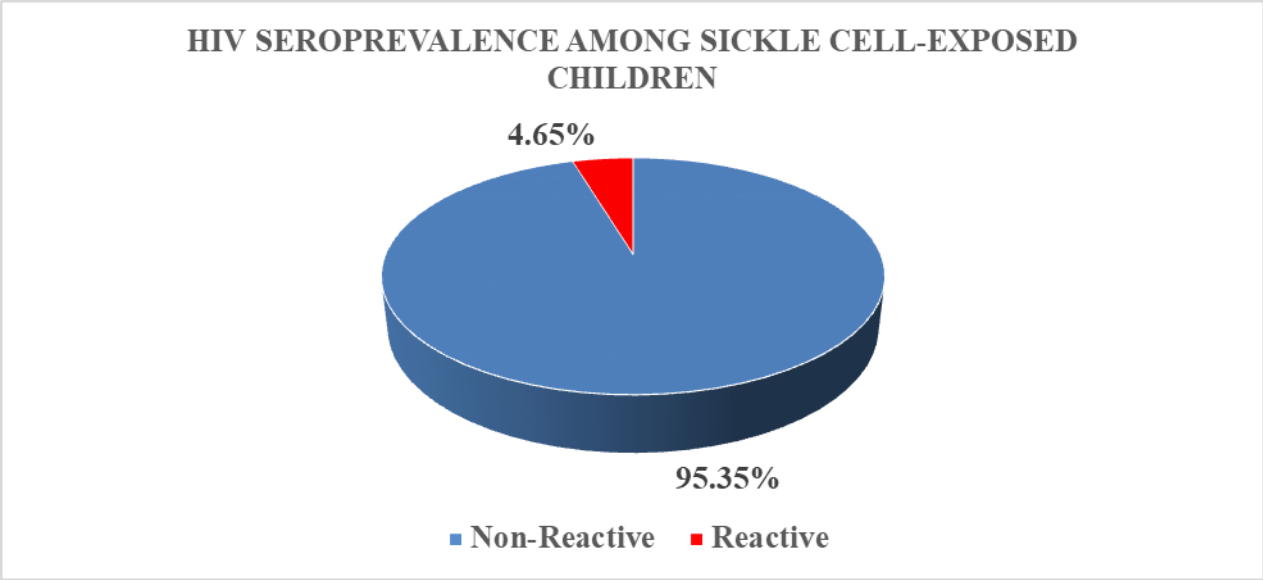

Fig.4 :HIV Seroprevalence in Sickle Cell-Exposed Children

In terms of gender, (see table 3 below), more male children (54.6%, n=82) were sickle cell-exposed compared to female children who were sickle cell-exposed (45.33%, n=68). However, co-infection was higher in female children than the male children.

Table 3: HIV Seroprevalence among Children by Gender and Age (Qualitative)

Variables	**N (%)**	**Non Reactive n (%)**	**Reactive n (%)**
Gender			
Female	68(45.33)	63(92.65)	5(7.35)
Male	82(54.67)	80(97.56)	2(2.44)
Age (Months)			
<6	8(4.65)	8(100)	0(0)
7 to 19	47(27.33)	45(95.74)	2(4.26)
20 to 32	20(11.63)	20(100)	0(0)
33 to 45	30(17.44)	29(96.67)	1(3.33)
46 to 58	18(10.47)	16(88.89)	2(11.11)
59 to 71	11(6.4)	9(81.82)	2(18.18)
72 to 84	7(4.07)	7(100)	0(0)
85 to 97	1(0.58)	1(100)	0(0)
98 to 110	8(4.65)	7(87.5)	1(12.5)
111 to 123	5(2.91)	5(100)	0(0)
> 123	17(9.88)	17(100)	0(0)
All Ages	172 (100)	164(95.35)	8(4.65)

None of the children below 6 months tested reactive for HIV.more female children who were sickle cell-exposed tested reactive for HIV (7.35%, n=5). Only (2.44%, n=2) of the sickle cell-exposed male children positive for co-infection. The age group with highest proportion testing reactive for HIV among sickle cell-exposed children was 59 months to 71 months (18.8%, n=2) . Other notable proportions were age group 98 months to 110 months (12.5%, n=1), 46 months to 58 months (11.11%, n=1). Age group 7 months to 19 months was (4.26%, n=2) with the co-infection.

Figure 5 below is bar graph showing HIV Seroprevalence across the age brackets by Gender and viral load. HIV viral load according to gender and age among the sickle cell-exposed children. Sickle cell-exposed male children who were also HIV positive were between the age 7 months to 19 months. Majority (n=2) of sickle

cell-exposed female children were between the age 46 months to 58 months. Other sickle cell-exposed female children were in the cohorts of 7 months to 19 months, 33 months to 45 months, and 59 months to 71 months.

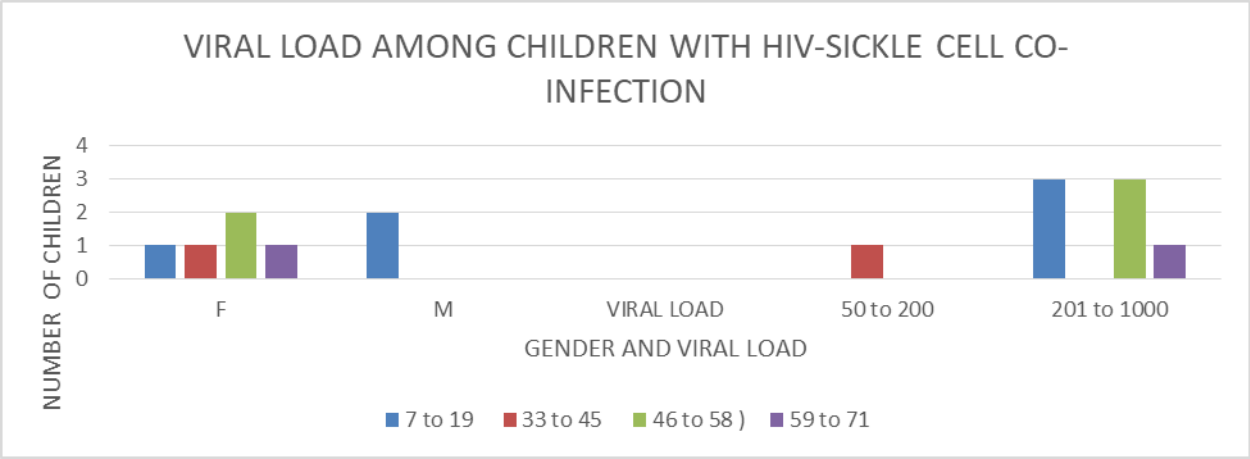

Fig. 5: Bargraph showing viral load status among HIV/AIDS and Sickle cell-exposed children

On the other end, minority of the sickle cell-exposed children with co-infection had 50 to 200 copies per ml and were between 33 months to 45 months. Majority of the sickle cell-exposed children with co-infection had 201 to 500 copies per ml and were between the age 7 months to 19 months and 46 months to 58 months. More than three quarters (75%, n=6) co-infected children were in two cohorts: 7 months to 19 moths and 46 months to 58 months and a viral load between 201 to 500 copies per ml of whole blood.

4.3 Association of HIV and hemoglobin types among sickle cell-exposed children

The association between gender and HIV-seropositivity had p-value=0.155. Age cohorts and reaction or non-reaction for HIV had p-value=0.414.

Table 4: Association between HIV and Sickle Cell-Exposed Children

Variables	N (%)	Non Reactive n (%)	Reactive n (%)	P-value
Gender				
Female	68(45.33)	63(92.65)	5(7.35)	0.155
Male	82(54.67)	80(97.56)	2(2.44)	
Age				
<6	8(4.65)	8(100)	0(0)	0.414
7 to 19	47(27.33)	45(95.74)	2(4.26)	
20 to 32	20(11.63)	20(100)	0(0)	
33 to 45	30(17.44)	29(96.67)	1(3.33)	
46 to 58	18(10.47)	16(88.89)	2(11.11)	
59 to 71	11(6.4)	9(81.82)	2(18.18)	
72 to 84	7(4.07)	7(100)	0(0)	
85 to 97	1(0.58)	1(100)	0(0)	
98 to 110	8(4.65)	7(87.5)	1(12.5)	
111 to 123	5(2.91)	5(100)	0(0)	
> 123	17(9.88)	17(100)	0(0)	
All Ages	172(100)	164(95.35)	8(4.65)	
SC-STATUS				0.937
S	131(76.16)	125(95.42)	6(4.58)	
T	41(23.84)	39(95.12)	2(4.88)	

Table 5 below shows that the association of viral load age cohort among sickle cell-exposed children had p-value= 0.046. Gender, and sickle cell status had p-values= 0.292, and 0.217 respectively.

Table 5: Association between Gender, viral load, sickle cell exposure status and age cohort among Sickle Cell-Exposed Children

Variables	7 to 19 n(%)	33 to 45 n(%)	46 to 58 n(%)	59 to 71 n(%)	P-value
Gender					
F	1(20)	1(20)	2(40)	1(20)	0.292
M	2(100)	0(0)	0(0)	0(0)	
HIV_CMLS					
50 to 200	0(0)	1(100)	0(0)	0(0)	**0.046**
201 to 1000	3(42.86)	0(0)	3(42.86)	1(14.29)	
HIV_STATUS					
R	3(37.5)	1(12.5)	3(37.5)	1(12.5)	
SC STATUS					
S	2(33.33)	1(16.67)	3(50)	0(0)	0.217
T	1(50)	0(0)	0(0)	1(50)	

In the bivariate logistic regression, the male sickle cell-exposed children were 0.4 times less likely to develop complication from the HIV co-infection among sickle cell-exposed children, p-value = 0.02. The sickle cell trait children were 3.85 times more likely to develop complications from HIV co-infection among sickle cell-exposed children, p-value= 0.001.

Table 6: Bivariate Logistic Regression of Gender, Viral Load, Sickle Cell status, and Hemoglobin Type among Sickle Cell-Exposed Children

Variables	COR(95% CI)	P-Value
GENDER		
F	**REF**	
M	0.40(0.19-0.86)	**0.02**
HIV STATUS		
N	**REF**	
R	1.69(0.39-7.37)	0.486
SC EXPOSURE		
S	**REF**	
T	3.85(1.82-8.13)	**<0.001**

Multivariate logistic analysis however showed that the male sickle cell-exposed children were 0.42 times less likely to develop complications from HIV co-infection among sickle cell-exposed children, p-value= 0.039.

Table 7: Multivariate Logistic Regression of Gender, Viral Load, Sickle Cell status, and Hemoglobin Type among Sickle Cell-Exposed Children

Variables	AOR(95% CI)	P-Value
GENDER		
FEMALE	**REF**	
MALE	0.42(0.18-0.96)	**0.039**

Discusssion and Interpretation

5.1 Introduction

This chapter discussed the findings per specific objectives. It also compared previous study findings to the findings of this study. The discussions were presented sequentially on the two specific objectives which were to establish the seroprevalence of HIV among sickle cell-exposed children in Kisumu County, and to determine the influence of HIV co-infection among sickle cell-exposed children in Kisumu County.

5.2 Seroprevalence of HIV among sickle cell-exposed children

The findings of this study provide valuable insights into the epidemiology of HIV in this vulnerable population and contribute to the growing body of literature on HIV-Sickle Cell co-infection..

The seroprevalence of HIV among sickle cell-exposed children was 4.65% (n=8). This indicated that approximately 5 out of 100 sickle cell-exposed children are infected and affected by HIV infection in Kisumu County. This prevalence rate is consistent with previous research by Owusu *et. al.*,(2014) who undertook a retrospective systematic review in Sub-Saharan Africa region. His findings were that HIV co-infection among sickle cell children was between 0% to 11.5% and overlapping in different Countries within the region of study. It is notable that there was similarity in study design: retrospective and the setting was also similar being the Low and Middle-Income Countries. According to Okafor *et al.,* (2012), HIV co-infection among children in Sub-Saharan Africa region was between 5% -15%. This was slightly above the findings of this study.

In Enugu Nigeria, Ubesie *et al*.,(2012) undertook a case-control study design on children with sickle cell infection. The cases were those children with sickle cell disease who had monotransfusion of blood, while the controls were those children with sickle cell who were not given any blood transfusion. He found out that 2.9% of the children with sickle cell given blood transfused acquired HIV infection. About 1.6% of children with sickle cell infection who had no blood transfusion had HIV co-infection. Another researcher Baah *et.al.,* 2014, found the prevalence of HIV among sickle cell patients as 6.7%. This study was done in Cape Coast, Ghana. The study adopted a cross-sectional study design. This is within the acceptable +_5% margin of error as outlined in this study and therefore also consistent with the finding of this study of 4.65% HIV co-infection among sickle cell-exposed children. In Southwestern Nigeria, Odaibo *et al.,* (2021) had varied results. His findings were that the HIV prevalence was higher in controls non-transfused children as opposed to the cases transfused children with sickle cell disease at 1.1% and 0.2% respectively. This was lower than the findings of this study though within the margin of error 5%. A study done in Yaounde Central Hospital in Ghana by Sack *et al.,* (2013) under Health Sciences and Disease found that 5.6% of sickle cell children were seropositive for HIV. This again was only 1% more than the findings of this study. In 2005, Ahmadu Bello University Teaching Hospital in Zaria Nigeria used retrospective study design to determine the prevalence of HIV among sickle cell children. The parents of the children were all seronegative for HIV while 45.5% of the children were positive for blood transfusion. The findings of this study indicated a 1.8% HIV seropositivity of the children.

Gender disparities were evident in the prevalence of HIV among sickle cell-exposed children, with a higher rate of co-infection observed among female children compared to male children. This finding is consistent with previous studies that have reported gender differences in HIV prevalence among various populations (AlZahrani *et al.,* 2020). However, the underlying factors contributing to this gender disparity in HIV prevalence among sickle cell-exposed children warrant further investigation. Age emerged as a significant factor influencing HIV seroprevalence among sickle cell-exposed children, with older age groups exhibiting higher rates of co-infection. This finding aligns with previous research indicating that older individuals are often more vulnerable to HIV infection due to factors such as polytransfusion of blood, increased mobility, social interactions, and sexual activity (Ballas *et al.,* 2012). Furthermore, the age distribution of HIV-positive cases among sickle cell-exposed children highlights the need for age-specific interventions and targeted outreach efforts to prevent new infections and improve health outcomes.

The study aimed to characterize the hemoglobin types among sickle cell-exposed children. The findings revealed that 76.16% (n=131) of the children had HB S suffering sickle cell disease. This implied that the over three quarters of the children had more than 41% of the abnormal hemoglobin S and less than 60% of the normal hemoglobin A0 of all the red blood cell hemoglobin types as determined through hemoglobin electrophoresis.
The finding also revealed that 23.84% (n=41) had sickle cell trait status implying that over a fifth of the children had less than 41% of the abnormal hemoglobin S and between 60% to 95% of the normal hemoglobin A0.

According to S. Colaco *et al.* (2022) the percentage proportion of children with sickle cell disease among sickle cell-exposed children was 72.5%. The proportion was slightly lower than the finding in this study result but within the margin of error 5%. The cross-sectional experimental study was carried out in Mumbai, India. In another study by SAA Khaled *et al.* (2022) in Egypt, the study which was a case-control design found a slightly higher rate of 78.2% for sickle cell disease among sickle cell-exposed children. The results of this study is therefore consistent with previous studies in terms of the high prevalence of sickle cell disease among exposed children. The proportion of the sickle cell train remains a but a fifth of the sickle cell-exposed children.

When considering the distribution of sickle cell disease among different age groups, this study found that the age cohort between 7 months to 19 months had the highest number of affected children. Specifically, 29.01% (n=38) of the children in this age group had sickle cell disease. This is similar to the findings of DA Vargas-Hernandez *et al*. (2023), who also found that the highest prevalence of sickle cell disease was in children aged 7 to 19 months, with a prevalence rate of 31.7%. In contrast, B. Kingchaiyaphum *et al.* (2020) reported a lower prevalence rate of 18.6%.

5.3 Association between HIV seroprevalence and Hemoglobin types among sickle cell-exposed children

The association between gender and HIV seropositivity had a p-value of 0.155. This suggests that there was no significant association between gender and HIV seropositivity among sickle cell-exposed children. Previous study by BJ Bain *et al.* (2023), S. Colaco *et al.* (2022), and SAA Khaled *et al*. (2022) also reported non-significant associations between gender and HIV seropositivity among similar populations.

The association between age cohorts and HIV seropositivity had a p-value of 0.414. This suggests that there is no significant association between age and HIV seropositivity among sickle cell-exposed children. Similar findings were reported by BJ Bain *et al*. (2023) and Owusu *et al.* (2015) who indicated no significant association between age and HIV seropositivity.

The association between gender and viral load had p-value of 0.047, This suggested a significant association between gender and viral load among sickle cell-exposed children. This finding was convergent with the results of S. Colaco et al. (2022), who reported a significant association between gender and HIV seropositivity (p-value < 0.05). The association between age cohorts and viral load had a p-value of 0.134, suggesting that there was no significant association between age and viral load among sickle cell-exposed children. As contrary to the finings herein, Owusu *et al.* (2015), reported a significant association between age and HIV seropositivity (p-value < 0.05). among sickle cell-exposed children.

The association between gender and age cohort had a p-value of 0.292, suggesting insignificant association between gender and age among sickle cell-exposed children.Similarly, S. Colaco *et al.* (2022) reported a non-significant association between gender and age cohort among similar populations (p-value > 0.05). Age cohort and viral load had a p-value of 0.134, suggesting that there was no significant association between age cohort and viral load among sickle cell-exposed children. This finding contradicted the results of DA Vargas-Hernandez *et al.* (2023), who reported a significant association between age cohort and HIV seroprevalence (p-value < 0.05).

There were little information on previous studies directly comparing age cohort and HBF among this population. The association between sickle cell status and age cohort had a p-value of 0.217.
This suggested that there was no significant association between sickle cell status and age cohort among sickle cell-exposed children. The association between gender and HIV viral load had a p-value of 0.046. This suggested that there is a significant association between gender and HIV viral load among sickle cell-exposed children. However, this finding contrasted the results of S. Colaco *et al.* (2022), who reported a non-significant association between gender and HIV seropositivity (p-value > 0.05).

The association between sickle cell status and age cohort had a p-value of 0.217. This suggested that there is no significant association between sickle cell status and age cohort among sickle cell-exposed children. This finding was consistent with the results of Owusu *et al.* (2015), who also reported a non-significant association between sickle cell status and age cohort (p-value > 0.05).

In multiple logistic regression, the Odd Ratio was 0.42 (p-value =0.02), indicating that gender was statistically significant factor with male sickle cell-exposed children being 0.42 times less likely to be associated with complication of HIV complications due to HIV co-infection among sickle cell-exposed children. This finding was in divergent with the findings of S. Colaco *et al.* (2022) who reported a non-significant association between gender and sickle cell status among similar populations (p-value > 0.05). Odds Ration for sickle cell-status was 3.85. This suggest that sickle cell status was statistically significant with p-value = 0.001, implying that the children with sickle cell trait were 3.85 times more likely to be affected by HIV co-infection complications than their counter part children with sickle cell disease. These findings were divergent with the results of G. Ndeezi *et al.* (2016), who also reported a non-significant association between viral load and sickle cell status (p-value > 0.05).

The bivariate analysis indicates that male sickle cell-exposed children are 0.4 times less likely to be affected by HIV co-infection as compared to their female counterparts (p-value = 0.02). This suggests a protective effect of male gender against against complications of HIV co-infection. Multivariate analysis supported this finding, with male children being 0.42 times less likely to be affected by the HIV co-infection as opposed to their female sickle cell exposed counterparts (p-value = 0.039). This consistency between bivariate and multivariate analyses strengthens the evidence for a gender-related difference in susceptibility to HIV co-infection among sickle cell-exposed children.

AlZahrani *et al.* (2020) did not find a significant gender difference in their cohort. Similarly, studies by Ballas (2012). The current findings add to the literature by highlighting a potential gender difference that warrants further investigation.

Conclusion and Recommendations

6.1: Conclusion

This section makes conclusions on the two specific objectives which were to establish the seroprevalence of HIV among sickle cell-exposed children in Kisumu County, to determine the influence of HIV co-infection among sickle cell-exposed children in Kisumu County.

6.2 Seroprevalence of HIV among sickle cell-exposed children

The findings of this study provide valuable insights into the epidemiology of HIV in a vulnerable population of sickle cell-exposed children. The seroprevalence of HIV among sickle cell-exposed children was approximately 5 out of 100 of the sickle cell-exposed children in Kisumu County. Gender disparities were evident in the prevalence of HIV among sickle cell-exposed children, with a higher rate of co-infection observed among female children compared to male children. Age emerged as a significant factor influencing HIV seroprevalence among sickle cell-exposed children, with older age groups exhibiting higher rates of co-infection.

Majority of sickle cell-exposed children had sickle cell disease. Children aged 7 to 19 months had the highest prevalence of sickle cell disease. A substantial proportion of sickle cell-exposed children had significant concentration of hemoglobin S.

6.4 Influence of HIV Co-infection among sickle cell-exposed children

There were significant associations between gender, sickle cell status and HIV viral load. Sickle cell trait was found to be having a protective effect as compared to sickle cell disease.

6.5 Recommendations

1) **Objective One: Seroprevalence of HIV among sickle cell-exposed children**

a) Establish targeted HIV prevention and education programs specifically tailored to sickle cell-exposed children, focusing on vulnerable groups such as female children and older age cohorts. Allocate resources to support comprehensive HIV screening and treatment services within healthcare facilities in areas with high prevalence rates of sickle cell disease.

b) Collaborate with healthcare providers and community-based organizations to develop and implement age-specific and gender-specific HIV prevention and education initiatives. Provide training and

resources to healthcare workers to improve their capacity for HIV screening, diagnosis, and treatment among sickle cell-exposed children.

c) Promote awareness about the risk of HIV among sickle cell-exposed children through community outreach programs, schools, and media campaigns. Encourage regular HIV testing and early intervention to reduce the burden of HIV among this vulnerable population.

Objective three: Association between HIV co-infection among sickle cell-exposed children

a) Support further research to better understand the relationship between hemoglobin types and HIV outcomes among sickle cell-exposed children. Allocate funding for longitudinal studies investigating the impact of hemoglobin types on HIV progression and treatment outcomes.

b) Collaborate with researchers, healthcare providers, and policymakers to develop comprehensive healthcare strategies for sickle cell-exposed children, considering the complex interplay between HIV co-infection among sickle cell-exposed children and HIV outcomes. Advocate for the integration of HIV screening and treatment services into existing sickle cell disease management programs.

c) Encourage individuals living with sickle cell disease to participate in HIV screening and treatment programs. Promote awareness about the importance of regular healthcare monitoring and adherence to treatment among sickle cell-exposed children.

6.6 Recommendation for further study

The following further study are recommended given the findings of this study.

a) Conduct longitudinal studies to investigate the trend of HIV progression, treatment outcomes, and overall health status among sickle cell-exposed children.

b) Explore the underlying mechanisms that may contribute to the observed associations in HIV co-infection.

c) Investigate how HIV co-infection influence immune function, disease progression, and response to antiretroviral therapy.

d) Explore the genetic factors that may predispose sickle cell-exposed children to both HIV infection and adverse health outcomes.

e) Investigate genetic variations that may modulate the risk of HIV seroprevalence and disease progression among individuals with different hemoglobin types.

f) Investigate the impact of co-infections, such as β-thalassemia, on HIV outcomes among sickle cell-exposed children.

g) Explore how co-existing hematological disorders may influence HIV seroprevalence, viral load, and disease progression.

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