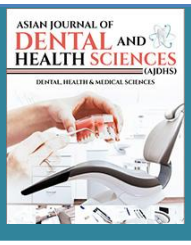

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Neurocognitive Impairment in HIV-Positive Sickle Cell Patients

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Abstract

Neurocognitive impairment is a significant yet often underappreciated complication in individuals co-infected with human immunodeficiency virus (HIV) and sickle cell disease (SCD). This review explores the interplay between these two conditions, highlighting the mechanisms that contribute to cognitive deficits. Chronic inflammation, vascular complications, and psychosocial stressors associated with both HIV and SCD create a unique vulnerability to neurocognitive decline, impacting daily functioning and overall quality of life. The prevalence of neurocognitive impairment in this population is concerning, with studies indicating that individuals with SCD may experience increased cognitive challenges due to the compounded effects of their condition and HIV-related factors. This article synthesizes current research on the prevalence, risk factors, and potential interventions for managing neurocognitive impairment in HIV-positive sickle cell patients. By identifying the unique challenges faced by this population, we can develop targeted therapeutic strategies aimed at preserving cognitive function and enhancing the quality of life.

Keywords: Neurocognitive Impairment, HIV, Sickle Cell Disease, Inflammation, Cognitive Function

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Introduction

Neurocognitive impairment has emerged as a significant concern in the context of chronic illnesses, particularly among individuals living with human immunodeficiency virus (HIV) and sickle cell disease (SCD). Both conditions independently contribute to a range of complications, including neurological deficits; however, their co-occurrence presents a unique clinical challenge. With the advent of effective antiretroviral therapies, the life expectancy of HIV-positive individuals has improved dramatically, leading to an increasing population of patients facing the dual burden of HIV and SCD. This demographic shift necessitates a comprehensive understanding of the neurocognitive implications associated with these concurrent conditions.[1-2] HIV-associated neurocognitive disorder (HAND) encompasses a spectrum of cognitive impairments, from mild deficits to severe dementia, affecting a substantial proportion of HIV-infected individuals. In patients with SCD, neurocognitive issues can arise due to a variety of factors, including recurrent pain episodes, silent cerebral infarcts, and other vascular complications that compromise cerebral blood flow. The intersection of these two conditions complicates the clinical picture, as individuals may experience compounded cognitive challenges that significantly impact their daily functioning, educational attainment, and overall quality of life.[3-4] Chronic inflammation is a key feature in both HIV and SCD, contributing to neurocognitive impairment through various mechanisms. In HIV, the persistent inflammatory response associated with viral replication can lead to neuroinflammation, disrupting neuronal signaling and promoting neurodegeneration. In patients with SCD, hemolysis and the subsequent release of inflammatory mediators can similarly impact brain health. This shared inflammatory milieu exacerbates cognitive decline in individuals with both conditions, underscoring the need for a deeper exploration of the interplay between inflammation and neurocognitive function.[5-6]

Moreover, vascular complications are prevalent in both HIV and SCD, further increasing the risk of cognitive impairment. In SCD, episodes of vaso-occlusion can lead to ischemic damage and silent cerebral infarcts, which are often asymptomatic but can have long-term consequences for cognitive function. HIV-associated changes in cerebral vasculature, including inflammation and endothelial dysfunction, can similarly impair blood flow to critical brain regions. Understanding how these vascular changes contribute to neurocognitive decline is crucial for developing targeted interventions that address the unique needs of this population.[7-8] Psychosocial factors also play a significant role in neurocognitive impairment among HIV-positive sickle cell patients. The chronic stress associated with managing multiple health conditions, coupled with

social determinants of health such as socioeconomic status and access to healthcare, can exacerbate cognitive decline. Anxiety, depression, and social isolation are common in this patient population, further impacting cognitive function and quality of life. Addressing these psychosocial aspects is essential for a holistic approach to patient care and cognitive health.[9-10] Assessing neurocognitive function in this complex population requires a comprehensive approach that considers both clinical and contextual factors. Standardized neuropsychological assessments, along with qualitative measures such as patient-reported outcomes and caregiver observations, can provide valuable insights into cognitive functioning. Early identification of cognitive deficits allows for timely intervention, which is critical for preserving cognitive health and improving overall patient outcomes.[11-12]

Epidemiology of Neurocognitive Impairment in HIV and SCD

The epidemiology of neurocognitive impairment among individuals with human immunodeficiency virus (HIV) and sickle cell disease (SCD) is complex and multifactorial, with both conditions contributing to an increased risk of cognitive deficits. Studies have shown that neurocognitive impairment is prevalent in both populations, but the co-occurrence of HIV and SCD further exacerbates this issue. In HIV-positive individuals, it is estimated that between 30% and 50% exhibit some degree of cognitive dysfunction, ranging from mild cognitive impairment to more severe forms of HIV-associated neurocognitive disorder (HAND). This impairment can manifest as difficulties in attention, memory, executive function, and psychomotor speed, significantly affecting the individual's quality of life and daily functioning.[13-15] Similarly, SCD is associated with an elevated risk of neurocognitive deficits, primarily due to the disease's complications, such as recurrent pain episodes, silent cerebral infarcts, and cerebrovascular accidents. Studies indicate that approximately 20% to 30% of children and adults with SCD experience cognitive impairments, with a higher prevalence observed in those with a history of cerebrovascular events. Neuroimaging studies have revealed that silent cerebral infarcts are common in individuals with SCD, occurring in up to 50% of affected patients, which can lead to subtle yet significant cognitive declines. The cumulative effects of recurrent pain, hypoxia, and vascular occlusion further increase the risk of cognitive impairment in this population.[16-17]

When considering the combined effects of HIV and SCD, the prevalence of neurocognitive impairment is even more pronounced. Research indicates that individuals co-infected with HIV and SCD experience a higher incidence of cognitive deficits compared to those with either condition alone. This heightened risk may be attributed to the overlapping pathophysiological mechanisms of both diseases, including chronic inflammation, oxidative stress, and vascular dysfunction. Additionally, the psychosocial stressors associated with managing two chronic conditions can contribute to cognitive decline, further complicating the epidemiological landscape.[18-19] Demographic factors also play a significant role in the prevalence of neurocognitive impairment among individuals with HIV and SCD. Age, sex, and socioeconomic status can influence cognitive outcomes, with older individuals and those from lower socioeconomic backgrounds at greater risk for cognitive deficits. Furthermore, access to healthcare and effective treatment options can impact the management of both HIV and SCD, potentially influencing the degree of neurocognitive impairment observed in these populations.[20]

Mechanisms of Neurocognitive Impairment in HIV-Positive Sickle Cell Patients

Neurocognitive impairment in individuals co-infected with human immunodeficiency virus (HIV) and sickle cell disease (SCD) arises from a complex interplay of biological, neurological, and psychosocial mechanisms.

1. Chronic Inflammation:

Both HIV and SCD are associated with chronic inflammatory states that significantly contribute to neurocognitive impairment. In HIV, the persistence of viral replication leads to elevated levels of pro-inflammatory cytokines, which can disrupt neuronal function and promote neuroinflammation. This inflammatory milieu can result in neuronal apoptosis, synaptic dysfunction, and altered neurotransmitter signaling, all of which may impair cognitive processes. In SCD, recurrent vaso-occlusive crises and hemolysis contribute to inflammation, releasing inflammatory mediators that can further compromise brain health. The combined inflammatory burden in co-infected patients can exacerbate cognitive deficits, leading to more pronounced neurocognitive impairment.[21-22]

2. Vascular Dysfunction:

Cerebrovascular complications are prevalent in both HIV and SCD, contributing to the risk of neurocognitive impairment. In SCD, episodes of vaso-occlusion can cause ischemic damage and silent cerebral infarcts, leading to subtle cognitive declines that may accumulate over time. Similarly, HIV-related changes in vascular health, including endothelial dysfunction and increased vascular permeability, can compromise blood flow to the brain. The resultant hypoxia and nutrient deprivation can lead to neuronal injury and cognitive deficits. The intersection of these vascular complications in co-infected patients can significantly impact cerebral perfusion and overall cognitive function.[23]

3. Neuroanatomical Changes:

Neuroimaging studies have demonstrated structural changes in the brains of individuals with HIV and SCD. In HIV-positive individuals, findings may include cortical atrophy, white matter lesions, and changes in basal ganglia structure. These changes can disrupt normal brain function and contribute to cognitive impairment. In SCD, the presence of silent cerebral infarcts and alterations in brain morphology has been observed, correlating with cognitive deficits. In co-infected patients, these neuroanatomical alterations

may be compounded, leading to a more severe manifestation of cognitive impairment.[24]

4. Oxidative Stress:

Oxidative stress is another critical mechanism that contributes to neurocognitive impairment in this population. Both HIV and SCD are associated with increased oxidative stress due to the release of reactive oxygen species (ROS) from inflammatory cells and vascular dysfunction. Elevated levels of oxidative stress can lead to neuronal damage, mitochondrial dysfunction, and impaired synaptic plasticity. This oxidative burden can further exacerbate cognitive deficits, highlighting the need for interventions that target oxidative stress as a potential therapeutic strategy.[25]

5. Psychosocial Factors:

Psychosocial stressors significantly impact neurocognitive function among HIV-positive sickle cell patients. Chronic illness, stigma, and socioeconomic challenges can lead to heightened anxiety and depression, both of which are known to negatively affect cognitive performance. The interplay between mental health and cognitive function is particularly relevant in this population, as psychosocial stress can exacerbate the biological mechanisms contributing to neurocognitive impairment. Addressing these psychosocial factors through support systems and mental health interventions is essential for improving cognitive outcomes.[26-27]

Risk Factors for Cognitive Decline in HIV-Positive Sickle Cell Patients

Cognitive decline in individuals co-infected with human immunodeficiency virus (HIV) and sickle cell disease (SCD) is influenced by a myriad of risk factors that can exacerbate neurocognitive impairment.

1. Age:

Age is a significant risk factor for cognitive decline in both HIV and SCD populations. As individuals age, the natural decline in cognitive function can be compounded by the effects of chronic illnesses. Older adults with HIV are at increased risk of neurocognitive disorders due to the cumulative impact of HIV-related neurotoxicity and the aging process itself. In SCD, age-related changes in brain structure and function, coupled with a history of cerebrovascular events, further heighten the risk of cognitive impairment.[28]

2. Disease Severity:

The severity of both HIV and SCD can significantly influence cognitive outcomes. In HIV, factors such as high viral load, low CD4 counts, and advanced stages of disease are associated with increased risk of neurocognitive impairment. In SCD, individuals with a history of stroke, recurrent vaso-occlusive crises, and chronic pain are more likely to experience cognitive deficits. The cumulative burden of these conditions can exacerbate cognitive decline, making disease management critical in reducing risk.[29]

3. Inflammation and Immune Dysfunction:

Chronic inflammation and immune dysfunction play a pivotal role in cognitive decline among co-infected patients. Elevated levels of pro-inflammatory cytokines, common in both HIV and SCD, have been linked to neuroinflammation and neuronal damage. The persistent inflammatory response associated with HIV can lead to neurocognitive disorders, while the inflammatory milieu in SCD can exacerbate these effects. Individuals with both conditions may experience heightened inflammatory responses, increasing their risk of cognitive impairment.[30]

4. Socioeconomic Status and Access to Healthcare:

Socioeconomic factors significantly influence health outcomes and can exacerbate cognitive decline in HIV-positive sickle cell patients. Lower socioeconomic status is associated with limited access to healthcare, which can affect the management of both HIV and SCD. Individuals facing economic hardships may experience higher levels of stress, less engagement in health-promoting behaviors, and inadequate access to necessary medical interventions, all of which can contribute to cognitive decline. Additionally, education level may also play a role, as lower educational attainment is linked to poorer cognitive outcomes.[31]

5. Mental Health Conditions:

Mental health comorbidities, such as depression and anxiety, are prevalent among individuals with HIV and SCD, significantly impacting cognitive function. Psychological distress can exacerbate cognitive decline, as mental health issues can interfere with attention, memory, and executive functioning. In individuals co-infected with HIV and SCD, the interplay of chronic illness, psychosocial stressors, and mental health challenges creates a compounded risk for cognitive impairment. Addressing mental health concerns is essential for holistic patient care and cognitive health preservation.[32]

6. Lifestyle Factors:

Lifestyle factors, including diet, physical activity, and substance use, can also influence cognitive outcomes in this population. Poor nutrition and sedentary lifestyles are common among individuals with chronic illnesses, which can exacerbate inflammation and cognitive decline. Substance use, particularly alcohol and recreational drugs, can further impair cognitive function and interact negatively with medications used to manage HIV and SCD. Promoting healthy lifestyle choices is crucial for mitigating cognitive decline in co-infected patients.[33]

7. History of Cerebrovascular Events:

A history of cerebrovascular events, such as strokes or transient ischemic attacks (TIAs), significantly increases the risk of cognitive decline in both HIV and SCD populations. Silent cerebral infarcts are particularly concerning in SCD patients, often leading to undetected but progressive cognitive impairment. In HIV-positive individuals, cerebrovascular complications can result

from both the direct effects of the virus and secondary factors such as hypertension and hyperlipidemia. The presence of prior cerebrovascular events indicates a higher likelihood of subsequent cognitive decline.[34]

Assessment of Neurocognitive Function in HIV-Positive Sickle Cell Patients

The assessment of neurocognitive function in individuals co-infected with human immunodeficiency virus (HIV) and sickle cell disease (SCD) is crucial for identifying cognitive deficits and tailoring appropriate interventions. Given the complexities associated with both conditions, a comprehensive approach that incorporates various assessment tools and methodologies is essential for accurately evaluating neurocognitive function. Below are key components and considerations in the assessment process:

1. Clinical Evaluation:

The initial assessment of neurocognitive function typically begins with a thorough clinical evaluation, which includes taking a detailed medical history and performing a physical examination. This process helps identify any potential neurological symptoms, psychiatric comorbidities, and the severity of both HIV and SCD. Clinicians should gather information on the patient's cognitive concerns, daily functioning, and any changes in behavior or mood, which may provide valuable context for understanding their cognitive status.[35]

2. Standardized Neuropsychological Testing:

Standardized neuropsychological tests are essential tools for objectively assessing cognitive function in co-infected individuals. These tests evaluate various cognitive domains, including attention, memory, executive function, language, and visuospatial skills. Commonly used assessments include the Mini-Mental State Examination (MMSE), Montreal Cognitive Assessment (MoCA), and more extensive battery tests like the Wechsler Adult Intelligence Scale (WAIS) and the Wechsler Memory Scale (WMS). Administering these tests allows clinicians to quantify cognitive impairments and track changes over time, facilitating targeted interventions.[36]

3. Functional Assessments:

In addition to standardized testing, functional assessments are critical for understanding how cognitive deficits impact daily living activities. Instruments such as the Functional Independence Measure (FIM) and the Activities of Daily Living (ADL) scale evaluate an individual's ability to perform essential tasks, including self-care, mobility, and communication. Assessing functional outcomes provides insights into how cognitive impairments affect the patient's quality of life and independence, guiding rehabilitation efforts.[37]

4. Neuroimaging Techniques:

Neuroimaging techniques, such as magnetic resonance imaging (MRI) and computed tomography (CT), play a valuable role in the assessment of neurocognitive function. These imaging modalities can identify structural brain changes, such as cerebral atrophy, white matter lesions, and silent cerebral infarcts, which may contribute to cognitive impairment. In patients with SCD, neuroimaging can help detect changes related to ischemic damage, while in HIV-positive individuals, it can reveal the effects of the virus on brain structures. The integration of neuroimaging findings with cognitive assessments can enhance the understanding of the underlying mechanisms of impairment.[38]

5. Psychosocial and Behavioral Assessments:

Evaluating psychosocial factors is essential in understanding the broader context of neurocognitive function in co-infected individuals. Assessing mental health conditions, such as depression and anxiety, can provide insights into how these issues may affect cognitive performance. Tools like the Beck Depression Inventory (BDI) or the Hamilton Anxiety Rating Scale (HAM-A) can be used to quantify the impact of psychosocial factors on cognitive function. Additionally, assessing lifestyle factors, such as substance use and social support, can help identify areas that require intervention to improve cognitive health.[39]

6. Longitudinal Assessment:

Neurocognitive function should be assessed longitudinally to track changes over time, particularly in patients with chronic illnesses like HIV and SCD. Regular cognitive evaluations can help identify the onset of cognitive decline early, allowing for timely interventions. Longitudinal studies can also provide valuable information on the natural progression of cognitive impairment in co-infected individuals, informing treatment strategies and resource allocation.[40]

7. Multidisciplinary Approach:

Given the complexity of neurocognitive impairment in HIV-positive sickle cell patients, a multidisciplinary approach is essential for comprehensive assessment and management. Collaboration among healthcare providers, including neurologists, psychologists, social workers, and primary care physicians, ensures that all aspects of a patient's health are considered. This approach facilitates the development of personalized treatment plans that address both cognitive and psychosocial needs, ultimately improving patient outcomes.[41]

Potential Therapeutic Approaches for Neurocognitive Impairment in HIV-Positive Sickle Cell Patients

Addressing neurocognitive impairment in individuals co-infected with human immunodeficiency virus (HIV) and sickle cell disease (SCD) necessitates a multifaceted therapeutic approach. Given the complexity of both conditions and their impact on cognitive function, therapeutic strategies should aim to mitigate cognitive decline, manage comorbidities, and enhance overall quality of life. Here are several potential therapeutic approaches:

1. Antiretroviral Therapy (ART):

Effective antiretroviral therapy (ART) is crucial in managing HIV and preventing neurocognitive impairment. ART reduces viral load, improves immune function, and decreases inflammation, all of which are associated with better cognitive outcomes. Ensuring adherence to ART is essential, as non-adherence can lead to viral rebound and increased risk of neurocognitive decline. Regular monitoring of treatment efficacy and side effects can help optimize ART regimens for co-infected individuals, contributing to improved cognitive health.[42]

2. Hydroxyurea Therapy:

Hydroxyurea is a cornerstone treatment for sickle cell disease, primarily used to reduce the frequency of vaso-occlusive crises and associated complications. Research suggests that hydroxyurea may also have neuroprotective effects, potentially improving cognitive function in SCD patients. By increasing fetal hemoglobin levels and reducing hemolysis, hydroxyurea may mitigate the impact of sickle cell disease on brain health, thus reducing the risk of neurocognitive impairment.[43]

3. Cognitive Rehabilitation Therapy:

Cognitive rehabilitation therapy (CRT) involves structured interventions aimed at improving cognitive function and compensating for cognitive deficits. CRT can be tailored to the specific needs of HIV-positive SCD patients, focusing on areas such as attention, memory, and executive functioning. Techniques may include cognitive exercises, memory aids, and compensatory strategies that help individuals manage daily tasks despite cognitive impairments. Engaging in cognitive rehabilitation can enhance patients' coping skills and improve their overall quality of life.[44]

4. Psychosocial Interventions:

Addressing psychosocial factors is vital for managing neurocognitive impairment. Psychosocial interventions, including counseling and support groups, can help individuals cope with the emotional and psychological aspects of living with chronic illnesses. Strategies to reduce anxiety and depression, which are common in co-infected patients, can improve cognitive function and overall well-being. Additionally, fostering social support networks can provide emotional resilience, further aiding cognitive health.[45]

5. Lifestyle Modifications:

Encouraging healthy lifestyle modifications can play a significant role in preserving cognitive function. Interventions focusing on nutrition, physical activity, and sleep hygiene can help improve overall health and cognitive performance. A balanced diet rich in antioxidants, omega-3 fatty acids, and essential vitamins supports brain health. Regular physical activity has been shown to enhance cognitive function and reduce inflammation. Moreover, promoting good sleep hygiene is critical, as sleep disturbances are prevalent in both HIV and SCD populations and can negatively impact cognitive function.[46]

6. Pharmacological Interventions for Cognitive Symptoms:

In some cases, pharmacological interventions may be considered to address specific cognitive symptoms. Medications such as cholinesterase inhibitors or NMDA receptor antagonists may help improve cognitive function in individuals with neurocognitive disorders. However, careful evaluation and monitoring are necessary to assess the efficacy and potential side effects of these medications, particularly in patients with comorbid conditions.[47]

7. Neuroprotective Agents:

Exploring the use of neuroprotective agents could provide additional therapeutic avenues for mitigating cognitive impairment in co-infected patients. Agents that target inflammation, oxidative stress, and excitotoxicity may hold promise in preserving cognitive function. Research into compounds like antioxidants, anti-inflammatory agents, and neurotrophic factors could pave the way for novel treatment strategies aimed at protecting neuronal health in HIV-positive SCD patients.[48]

8. Multidisciplinary Care Approach:

Implementing a multidisciplinary care approach that integrates medical, psychological, and social services is essential for addressing the complex needs of co-infected individuals. Collaboration among healthcare providers, including hematologists, infectious disease specialists, neurologists, psychologists, and social workers, ensures comprehensive care that addresses both physical and cognitive health. This approach can facilitate early identification of cognitive deficits, timely interventions, and coordinated management of comorbidities, ultimately improving patient outcomes.[48]

Conclusion

Neurocognitive impairment in individuals co-infected with HIV and sickle cell disease (SCD) presents a significant challenge, impacting their quality of life and overall health outcomes. The intricate interplay between these two conditions necessitates a comprehensive understanding of the mechanisms underlying cognitive decline and the multifactorial approaches required for effective management. Through a combination of effective antiretroviral therapy, sickle cell management strategies, cognitive rehabilitation, psychosocial support, and lifestyle modifications, it is possible to mitigate the effects of neurocognitive impairment in this population. The assessment and management of neurocognitive function in HIV-positive SCD patients should adopt a multidisciplinary approach, incorporating the expertise of various healthcare providers. This collaboration enables a holistic perspective on patient care, addressing both the physical and cognitive aspects of their health. Ongoing research into novel therapeutic interventions, neuroprotective agents, and personalized care strategies will be essential in advancing our understanding and management of cognitive impairment in co-infected individuals.

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References

1. Owusu ED, Visser BJ, Nagel IM, Mens PF, Grobusch MP. The interaction between sickle cell disease and HIV infection: a systematic review. Clinical Infectious Diseases. 2015; 60(4):612-626. https://doi.org/10.1093/cid/ciu832 PMid:25344542

2. Boateng LA, Ngoma AM, Bates I, Schonewille H. Red blood cell alloimmunization in transfused patients with sickle cell disease in sub-Saharan Africa; a systematic review and meta-analysis. Transfusion Medicine Reviews. 2019; 33(3):162-169. https://doi.org/10.1016/j.tmrv.2019.06.003 PMid:31345590

3. Ola B, Olushola O, Ebenso B, Berghs M. Sickle Cell Disease and Its Psychosocial Burdens in Africa. InSickle Cell Disease in Sub-Saharan Africa 2024: 67-80. Routledge. https://doi.org/10.4324/9781003467748-7

4. Obeagu EI, Reducing Hospitalization Rates: The Preventive Benefits of Blood Transfusions in HIV Care, International Journal of Medical Sciences and Pharma Research, 2024;10(3):29-34 https://doi.org/10.22270/ijmspr.v10i3.111

5. Ochocinski D, Dalal M, Black LV, Carr S, Lew J, Sullivan K, Kissoon N. Life-threatening infectious complications in sickle cell disease: a concise narrative review. Frontiers in Pediatrics. 2020; 8:38. https://doi.org/10.3389/fped.2020.00038 PMid:32154192 PMCid:PMC7044152

6. Obeagu EI, Obeagu GU, Okwuanaso CB. Optimizing Immune Health in HIV Patients through Nutrition: A Review. Elite Journal of Immunology, 2024; 2(1): 14-33

7. Obeagu EI, Obeagu GU. Platelet Distribution Width (PDW) as a Prognostic Marker for Anemia Severity in HIV Patients: A Comprehensive Review. Journal home page: http://www.journalijiar. com.;12(01).

8. Obeagu EI, Ubosi NI, Obeagu GU, Akram M. Early Infant Diagnosis: Key to Breaking the Chain of HIV Transmission. Elite Journal of Public Health, 2024; 2 (1): 52-61

9. Obeagu EI, Obeagu GU. Hematocrit Fluctuations in HIV Patients Co-infected with Malaria Parasites: A Comprehensive Review. Int. J. Curr. Res. Med. Sci. 2024; 10(1):25-36. https://doi.org/10.22270/ijmspr.v10i2.95

10. Obeagu EI, Obeagu GU. Transfusion Therapy in HIV: Risk Mitigation and Benefits for Improved Patient Outcomes. Asian J Dental Health Sci, 2024; 4(1):32-7. https://doi.org/10.22270/ajdhs.v4i1.62

11. Obeagu EI, Obeagu GU. Advancements in HIV Prevention: Africa's Trailblazing Initiatives and Breakthroughs. Elite Journal of Public Health, 2024; 2 (1): 52-63

12. Obeagu EI, Obeagu GU. Optimizing Blood Transfusion Protocols for Breast Cancer Patients Living with HIV: A Comprehensive Review. Elite Journal of Nursing and Health Science, 2024; 2(2):1-17

13. Obeagu EI, Obeagu GU. Understanding ART and Platelet Functionality: Implications for HIV Patients. Elite Journal of HIV, 2024; 2(2): 60-73 1

14. Obeagu EI, Obeagu GU. Hematologic Considerations in Breast Cancer Patients with HIV: Insights into Blood Transfusion Strategies. Elite Journal of Health Science, 2024; 2(2): 20- 35

15. Obeagu EI, Obeagu GU. Impact of Maternal Eosinophils on Neonatal Immunity in HIVExposed Infants: A Review. Elite Journal of Immunology, 2024; 2(3): 1-18 https://doi.org/10.22270/ajdhs.v4i2.82

16. Obeagu EI, Obeagu GU, Obiezu J, Ezeonwumelu C, Ogunnaya FU, Ngwoke AO, Emeka-Obi OR, Ugwu OP. Hematologic Support in HIV Patients: Blood Transfusion Strategies and Immunological Considerations. Newport International Journal of Biological and Applied Sciences (NIJBAS) 2023. http://hdl.handle.net/20.500.12493/14626

17. Ntsekhe M, Baker JV. Cardiovascular disease among persons living with HIV: new insights into pathogenesis and clinical manifestations in a global context. Circulation. 2023; 147(1):83-100. https://doi.org/10.1161/CIRCULATIONAHA.122.057443 PMid:36576956

18. Obare LM, Temu T, Mallal SA, Wanjalla CN. Inflammation in HIV and its impact on atherosclerotic cardiovascular disease. Circulation research. 2024; 134(11):1515-1545 https://doi.org/10.1161/CIRCRESAHA.124.323891 PMid:38781301 PMCid:PMC11122788

19. Hmiel L, Zhang S, Obare LM, Santana MA, Wanjalla CN, Titanji BK, Hileman CO, Bagchi S. Inflammatory and immune mechanisms for atherosclerotic cardiovascular disease in HIV. International journal of molecular sciences. 2024; 25(13):7266. https://doi.org/10.3390/ijms25137266 PMid:39000373 PMCid:PMC11242562

20. Obeagu EI, Obeagu GU. Platelet Aberrations in HIV Patients: Assessing Impacts of ART. Elite Journal of Haematology, 2024; 2(3): 10-24

21. Obeagu EI, Obeagu GU. Harnessing B Cell Responses for Personalized Approaches in HIV Management. Elite Journal of Immunology, 2024; 2(2): 15-28

22. Belisário AR, Blatyta PF, Vivanco D, Oliveira CD, Carneiro-Proietti AB, Sabino EC, de Almeida-Neto C, Loureiro P, Máximo C, de Oliveira Garcia Mateos S, Flor-Park MV. Association of HIV infection with clinical and laboratory characteristics of sickle cell disease. BMC Infectious Diseases. 2020; 20(1):638. https://doi.org/10.1186/s12879-020-05366-z PMid:32854639 PMCid:PMC7457248

23. Obeagu EI, Addressing Sleep Disturbances: Blood Transfusions and Improved Sleep Patterns in HIV Patients, International Journal of Medical Sciences and Pharma Research, 2024;10(3):43-48 https://doi.org/10.22270/ijmspr.v10i3.113

24. Gill AF, Ahsan MH, Lackner AA, Veazey RS. Hematologic abnormalities associated with simian immunodeficieny virus (SIV) infection mimic those in HIV infection. Journal of Medical Primatology. 2012; 41(3):214-224. https://doi.org/10.1111/j.1600-0684.2012.00543.x PMid:22620272 PMCid:PMC3367385

25. Nouraie M, Nekhai S, Gordeuk VR. Sickle cell disease is associated with decreased HIV but higher HBV and HCV comorbidities in US hospital discharge records: a cross-sectional study. Sexually transmitted infections. 2012; 88(7):528-533. https://doi.org/10.1136/sextrans-2011-050459 PMid:22628662 PMCid:PMC3456988

26. Obeagu EI, Obeagu GU. Hematological Changes Following Blood Transfusion in Young Children with Severe Malaria and HIV: A Critical Review. Elite Journal of Laboratory Medicine. 2024; 2(1):33-45.

27. Obeagu EI, Obeagu GU. The Role of L-selectin in Tuberculosis and HIV Coinfection: Implications for Disease Diagnosis and Management. Elite Journal of Public Health, 2024; 2 (1): 35-51

28. Obeagu EI, Obeagu GU. Unraveling the Role of Eosinophil Extracellular Traps (EETs) in HIV-Infected Pregnant Women: A Review. Elite Journal of Nursing and Health Science, 2024; 2(3): 84-99

29. Obeagu EI, Obeagu GU. Unveiling the Role of Innate Immune Activation in Pediatric HIV: A Review. Elite Journal of Immunology, 2024; 2(3): 33-44

30. Obeagu EI, Obeagu, GU. Impact of Blood Transfusion on Viral Load Dynamics in HIVPositive Neonates with Severe Malaria: A Review. Elite Journal of Scientific Research and Review, 2024; 2(1): 42-60

31. Obeagu EI, Youth-Friendly HIV Prevention: Tailoring Interventions for Young Populations, International Journal of Medical Sciences and Pharma Research, 2024;10(4):62-67. https://doi.org/10.22270/ijmspr.v10i4.125

32. Obeagu EI, Faith-based initiatives, HIV awareness, religious communities, health education, stigma reduction, International Journal of Medical Sciences and Pharma Research, 2024;10(4):74-79 https://doi.org/10.22270/ijmspr.v10i4.127

33. Obeagu EI, Obeagu GU. P-Selectin Expression in HIV-Associated Coagulopathy: Implications for Treatment. Elite Journal of Haematology, 2024; 2(3): 25-41

34. Obeagu EI, Obeagu GU. P-Selectin and Immune Activation in HIV: Clinical Implications. Elite Journal of Health Science, 2024; 2(2): 16-29

35. Obeagu EI, Amaeze AA, Ogbu ISI, Obeagu GU. B Cell Deficiency and Implications in HIV Pathogenesis: Unraveling the Complex Interplay. Elite Journal of Nursing and Health Science, 2024; 2(2): 33-46

36. Obeagu EI, Obeagu, GU. Platelet Dysfunction in HIV Patients: Assessing ART Risks. Elite Journal of Scientific Research and Review, 2024; 2(1): 1-16

37. Kibaru EG, Nduati R, Wamalwa D, Kariuki N. Impact of highly active antiretroviral therapy on hematological indices among HIV-1 infected children at Kenyatta National Hospital-Kenya: retrospective study. AIDS research and therapy. 2015; 12:1-8. https://doi.org/10.1186/s12981-015-0069-4 PMid:26279668 PMCid:PMC4537535

38. Enawgaw B, Alem M, Addis Z, Melku M. Determination of hematological and immunological parameters among HIV positive patients taking highly active antiretroviral treatment and treatment naïve in the antiretroviral therapy clinic of Gondar University Hospital, Gondar, Northwest Ethiopia: a comparative cross-sectional study. BMC hematology. 2014; 14:1-7. https://doi.org/10.1186/2052-1839-14-8 PMid:24666771 PMCid:PMC3994311

39. Gudina A, Wordofa M, Urgessa F. Immuno-hematological parameters among adult HIV patients before and after initiation of Dolutegravir based antiretroviral therapy, Addis Ababa, Ethiopia. Plos one. 2024; 19(10):e0310239. https://doi.org/10.1371/journal.pone.0310239 PMid:39480901 PMCid:PMC11527299

40. Geletaw T, Tadesse MZ, Demisse AG. Hematologic abnormalities and associated factors among HIV infected children pre-and post-antiretroviral treatment, North West Ethiopia. Journal of blood medicine. 2017:99-105. https://doi.org/10.2147/JBM.S137067 PMid:28831276 PMCid:PMC5552149

41. Jegede FE, Oyeyi TI, Abdulrahman SA, Mbah HA, Badru T, Agbakwuru C, Adedokun O. Effect of HIV and malaria parasites co-infection on immune-hematological profiles among patients attending anti-retroviral treatment (ART) clinic in Infectious Disease Hospital Kano, Nigeria. PLoS One. 2017; 12(3):e0174233. https://doi.org/10.1371/journal.pone.0174233 PMid:28346490 PMCid:PMC5367709

42. Obeagu EI, Obeagu GU. ART and Platelet Dynamics: Assessing Implications for HIV Patient Care. Elite Journal of Haematology. 2024; 2(4):68-85.

43. Obeagu EI, Ayogu EE, Obeagu GU. Impact on Viral Load Dynamics: Understanding the Interplay between Blood Transfusion and Antiretroviral Therapy in HIV Management. Elite Journal of Nursing and Health Science. 2024;2(2):5-15.

44. Ciccacci F, Lucaroni F, Latagliata R, Morciano L, Mondlane E, Balama M, Tembo D, Gondwe J, Orlando S, Palombi L, Marazzi MC. Hematologic alterations and early mortality in a cohort of HIV positive African patients. PLoS One. 2020; 15(11):e0242068. https://doi.org/10.1371/journal.pone.0242068 PMid:33170905 PMCid:PMC7654783

45. Ashenafi G, Tibebu M, Tilahun D, Tsegaye A. Immunohematological Outcome Among Adult HIV Patients Taking Highly Active Antiretroviral Therapy for at Least Six Months in Yabelo Hospital, Borana, Ethiopia. Journal of Blood Medicine. 2023:543-554. https://doi.org/10.2147/JBM.S419414 PMid:37881654 PMCid:PMC10595970

46. Obeagu EI, Goryacheva OG. The Role of Inflammation in HIV and Sickle Cell Disease Co-Morbidity. Lifeline HIV, 2025; 3(1): 1-12

47. Obeagu EI, Goryacheva OG. Oxidative Stress in HIV and Sickle Cell Disease: A Double Burden. Lifeline HIV, 2025; 3(1): 13-24

48. Obeagu EI, Goryacheva OG. HIV and Sickle Cell Disease: A Focus on Liver Dysfunction. Lifeline HIV, 2025; 3(1): 25-40