Elite Journal of Haematology. Volume 2 issue 4(2024), Pp. 180-198
https://epjournals.com/journals/EJH

The Nexus Between Obesity and Leukemia Progression in HIV-Positive Individuals: A Review

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Abstract

The coexistence of obesity, leukemia, and HIV infection presents a multifaceted challenge in clinical management. This review aims to elucidate the intricate relationship between obesity and leukemia progression in individuals living with HIV. Obesity, characterized by chronic low-grade inflammation and metabolic dysregulation, influences leukemia progression through complex mechanisms involving adipose tissue dysfunction, altered immune responses, and systemic inflammation. Moreover, in HIV-positive individuals, the interplay between viral infection, immune dysfunction, and oncogenesis further exacerbates the impact of obesity on leukemia progression. Understanding these interactions is crucial for developing tailored therapeutic strategies to improve clinical outcomes in this vulnerable population.

Keywords: *Obesity, Leukemia, HIV, Progression, Adipose Tissue, Inflammation, Immune Dysregulation*

Introduction

The co-occurrence of obesity, leukemia, and HIV/AIDS represents a complex nexus of health challenges that profoundly impact individual health outcomes and public health systems globally. Obesity, a chronic condition characterized by excessive adipose tissue accumulation, is

Citation: Obeagu EI, Obeagu GU. The Nexus Between Obesity and Leukemia Progression in HIV-Positive Individuals: A Review**.** *Elite Journal of Haematology, 2024; 2(4):* 180-198

increasingly recognized as a significant risk factor for various malignancies, including leukemia. Concurrently, individuals living with HIV/AIDS face a heightened risk of developing leukemia due to the immunosuppressive effects of the virus and associated chronic inflammation. Understanding the intricate interplay between these three conditions is paramount for elucidating disease mechanisms and developing effective therapeutic strategies to improve clinical outcomes. Obesity, beyond its role as a metabolic disorder, is now acknowledged as a state of chronic low-grade inflammation, with adipose tissue serving as a dynamic endocrine organ secreting adipokines, cytokines, and other bioactive molecules. This adipose tissue dysfunction creates a pro-inflammatory microenvironment conducive to oncogenesis and tumor progression, including leukemia. The dysregulated secretion of adipokines and cytokines, such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), contributes to systemic inflammation, insulin resistance, and altered lipid metabolism, all of which foster leukemic cell survival and proliferation.[1-40]

In the context of HIV infection, the immune dysregulation and chronic inflammation associated with the virus further exacerbate the impact of obesity on leukemia progression. HIV-induced alterations in immune function, including CD4+ T-cell depletion and CD8+ T-cell expansion, disrupt immune surveillance mechanisms, allowing for the persistence of oncogenic viruses and leukemic transformation. Moreover, the dysregulation of cytokine networks in HIV/AIDS, characterized by elevated levels of pro-inflammatory cytokines and impaired antitumor immune responses, creates an environment conducive to leukemia development and progression. Antiretroviral therapy (ART), while essential for suppressing HIV replication and restoring immune function, may also influence leukemia progression through its immunomodulatory effects and metabolic disturbances. Understanding the complex interactions between obesity, HIV infection, and leukemia is crucial for optimizing treatment strategies and improving outcomes for individuals living with these comorbidities. This review aims to explore the multifaceted relationship between obesity and leukemia progression in HIV-positive individuals, providing insights into the underlying mechanisms and therapeutic implications to guide future research and clinical practice.[41-70]

Obesity and Leukemia Progression

Obesity, a multifaceted chronic condition characterized by excessive adipose tissue accumulation, has emerged as a significant factor influencing the progression of leukemia, a hematological malignancy characterized by abnormal proliferation of white blood cells. The relationship between obesity and leukemia progression is complex and involves a myriad of interconnected mechanisms spanning inflammation, metabolic dysregulation, and alterations in the bone marrow microenvironment. Central to the interplay between obesity and leukemia progression is the chronic low-grade inflammation characteristic of adipose tissue dysfunction. Adipose tissue serves not only as an energy storage depot but also as an active endocrine organ, secreting a multitude of bioactive molecules termed adipokines. Dysregulated secretion of adipokines, including pro-inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and

leptin, alongside decreased levels of anti-inflammatory adiponectin, creates a pro-inflammatory microenvironment conducive to leukemic cell survival and proliferation. Moreover, adipocytes and stromal cells within the adipose tissue niche interact with leukemia cells, promoting their migration, invasion, and resistance to chemotherapy-induced apoptosis.[71-100]

Metabolic dysregulation associated with obesity further contributes to leukemia progression through altered lipid metabolism and insulin resistance. Elevated levels of circulating free fatty acids and dyslipidemia can directly impact leukemic cell behavior and chemoresistance. Additionally, insulin resistance and hyperinsulinemia, common features of obesity, have been implicated in promoting leukemic cell growth and survival through activation of insulin-like growth factor-1 (IGF-1) signaling pathways. The bone marrow microenvironment, essential for hematopoiesis and leukemic cell homing, is profoundly influenced by obesity. Adipose tissue expansion and adipocyte infiltration into the bone marrow disrupt the balance of hematopoietic stem cell niches, favoring the expansion of leukemic stem cells and disease progression. Adipocyte-derived factors, including adipokines, extracellular vesicles, and fatty acids, modulate signaling pathways involved in leukemic cell growth, survival, and chemoresistance. While the precise mechanisms linking obesity and leukemia progression continue to be elucidated, the clinical implications are clear. Obesity represents a modifiable risk factor that may influence leukemia prognosis and treatment response. Therefore, interventions targeting obesity-associated inflammation and metabolic dysregulation hold promise for improving outcomes in leukemia patients, particularly those with comorbid obesity. Further research is warranted to explore the intricate interplay between obesity and leukemia progression and to develop targeted therapeutic strategies aimed at disrupting this detrimental association.[101-140]

Role of HIV Infection

The role of HIV infection in the progression of leukemia, particularly in the context of obesity, adds another layer of complexity to the disease process. HIV/AIDS, a viral infection characterized by progressive immunosuppression and chronic inflammation, significantly influences the pathogenesis and clinical course of leukemia in affected individuals. One of the primary mechanisms through which HIV infection contributes to leukemia progression is immune dysregulation. The virus targets CD4+ T cells, leading to their depletion and impairing immune surveillance mechanisms. This immune dysfunction compromises the host's ability to recognize and eliminate malignant cells, allowing for the proliferation and dissemination of leukemic clones. Furthermore, HIV-induced alterations in T-cell subsets, including an increase in CD8+ T cells and a decrease in CD4+/CD8+ ratio, disrupt immune regulation and antitumor immunity, fostering a microenvironment favorable for leukemia development and progression. Chronic inflammation associated with HIV/AIDS further exacerbates leukemia progression by creating a pro-inflammatory milieu that promotes leukemic cell survival and proliferation. HIV-infected individuals often exhibit elevated levels of inflammatory cytokines, such as interleukin-6 (IL-6), interleukin-10 (IL-10), and tumor necrosis factor-alpha (TNF-α), which contribute to

leukemogenesis and disease progression. Additionally, HIV-associated co-infections with oncogenic viruses, such as Epstein-Barr virus (EBV) and human T-cell lymphotropic virus type 1 (HTLV-1), further increase the risk of leukemia development through viral oncogene expression and immune evasion mechanisms. Antiretroviral therapy (ART), the cornerstone of HIV management, has revolutionized the treatment landscape and significantly improved survival rates among HIV-positive individuals. However, the impact of ART on leukemia progression remains a subject of debate. While ART effectively suppresses viral replication and restores immune function, certain antiretroviral drugs may exert direct or indirect effects on leukemic cells, influencing disease progression and treatment outcomes. Additionally, metabolic side effects of ART, such as dyslipidemia and insulin resistance, may further contribute to leukemia progression in individuals with comorbid obesity.[141-200]

Conclusion

The convergence of obesity, HIV infection, and leukemia presents a multifaceted challenge in clinical management, necessitating a comprehensive understanding of their complex interactions. Obesity, characterized by chronic low-grade inflammation and metabolic dysregulation, contributes to leukemia progression through adipose tissue dysfunction, altered immune responses, and systemic inflammation. Similarly, HIV infection, with its immunosuppressive effects and chronic inflammation, further exacerbates leukemia progression by impairing immune surveillance mechanisms and creating a pro-inflammatory microenvironment conducive to leukemogenesis.

The interplay between obesity, HIV infection, and leukemia underscores the importance of personalized therapeutic approaches tailored to the specific needs of affected individuals. Targeted interventions aimed at mitigating chronic inflammation, restoring immune function, and addressing metabolic dysregulation hold promise for improving clinical outcomes in this vulnerable population. Furthermore, elucidating the molecular mechanisms underlying these interactions will facilitate the development of novel therapeutic strategies to disrupt the detrimental association between obesity, HIV infection, and leukemia progression.

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Citation: Obeagu EI, Obeagu GU. The Nexus Between Obesity and Leukemia Progression in HIV-Positive Individuals: A Review**.** *Elite Journal of Haematology, 2024; 2(4):* 180-198