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Cytotoxic T-Lymphocyte-Associated Protein 4 (CTLA-4) Blockade and HIV-Associated Kaposi Sarcoma: A Promising Therapeutic Strategy

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Abstract

Kaposi sarcoma (KS) remains a significant oncologic complication of HIV infection, particularly in regions with high HIV prevalence. Despite advances in antiretroviral therapy (ART), the incidence of KS remains elevated among HIV-infected individuals, highlighting the need for novel therapeutic approaches. CTLA-4, a key immune checkpoint molecule, plays a crucial role in regulating T cell activation and immune tolerance. Recent studies have implicated dysregulated CTLA-4 signaling in the pathogenesis of KS, suggesting that CTLA-4 blockade may represent a promising therapeutic strategy for this malignancy. In this review, we discuss the role of CTLA-4 in KS pathogenesis, preclinical and clinical evidence supporting CTLA-4 blockade as a therapeutic approach for HIV-associated KS, and potential challenges and future directions in this field.

Keywords: *CTLA-4, immune checkpoint blockade, Kaposi sarcoma, HIV/AIDS, immunotherapy*

Introduction

Kaposi sarcoma (KS) has long been recognized as one of the hallmark malignancies associated with HIV/AIDS. Despite advancements in antiretroviral therapy (ART), KS continues to pose a significant clinical challenge, particularly in resource-limited settings and among immunocompromised individuals. Its etiology is closely linked to infection with human herpesvirus-8 (HHV-8), also known as Kaposi sarcoma-associated herpesvirus (KSHV), which establishes a lifelong infection and can lead to the development of KS under conditions of immunosuppression. The introduction of ART has led to a substantial reduction in the incidence of KS; however, it remains prevalent, especially in individuals with advanced HIV disease and

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those with suboptimal access to healthcare services. CTLA-4, a critical immune checkpoint molecule, has garnered increasing attention in cancer immunotherapy due to its role in regulating T cell activation and immune tolerance. In recent years, dysregulated CTLA-4 signaling has been implicated in the pathogenesis of various malignancies, including melanoma, lung cancer, and lymphoma. Given its role in immune regulation, CTLA-4 has emerged as a potential target for cancer immunotherapy, particularly immune checkpoint blockade, which aims to enhance antitumor immune responses by blocking inhibitory signals that suppress T cell activity.¹⁻³⁰

Preclinical studies have provided compelling evidence supporting the efficacy of CTLA-4 blockade in promoting tumor regression and improving survival outcomes in various cancer models. Moreover, clinical trials investigating monoclonal antibodies targeting CTLA-4, such as ipilimumab and tremelimumab, have demonstrated significant therapeutic benefits in patients with advanced melanoma, leading to their approval by regulatory agencies for clinical use. The success of CTLA-4 blockade in other malignancies has sparked interest in exploring its potential application in HIV-associated KS, where immune dysregulation and impaired T cell function play a crucial role in tumor progression. Despite the promising prospects of CTLA-4 blockade in HIV-associated KS, several challenges and unanswered questions remain. These include identifying predictive biomarkers to select patients likely to benefit from therapy, optimizing treatment regimens to minimize toxicities, and elucidating the impact of CTLA-4 blockade on HIV-specific immune responses and viral control. Addressing these issues will be essential for translating preclinical and early clinical findings into effective therapeutic strategies for HIV-infected individuals with KS, ultimately improving clinical outcomes and quality of life in this population.³¹⁻⁶⁰

CTLA-4 Signaling in KS Pathogenesis

CTLA-4 (cytotoxic T-lymphocyte-associated protein 4) signaling plays a pivotal role in the pathogenesis of Kaposi sarcoma (KS), a multifocal vascular tumor associated with human herpesvirus-8 (HHV-8) infection. As an immune checkpoint molecule, CTLA-4 regulates T cell activation and tolerance, exerting a significant influence on the immune response against HHV-8-infected cells and the development of KS lesions. HHV-8 infection induces alterations in the host immune response, including the upregulation of CTLA-4 expression on T cells. This upregulation may contribute to T cell exhaustion and dysfunction, impairing the antiviral immune response and allowing for viral persistence and KS tumor growth. Additionally, HHV-8-encoded proteins, such as viral interferon regulatory factor (vIRF)-1, have been shown to enhance CTLA-4 expression on infected cells, further facilitating immune evasion and KS pathogenesis. Within KS lesions, increased CTLA-4 expression has been observed on both tumor-infiltrating lymphocytes and antigen-presenting cells, indicating its potential role in modulating the local immune microenvironment. CTLA-4 signaling may inhibit effector T cell responses against HHV-8-infected cells and dampen antitumor immunity, thereby promoting KS tumor growth and dissemination. Furthermore, CTLA-4 expression on regulatory T cells (Tregs) within KS lesions may contribute to immune suppression and tumor immune evasion, creating a favorable environment for KS development. The dysregulation of CTLA-4 signaling in KS pathogenesis

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suggests that targeting this immune checkpoint pathway could represent a promising therapeutic strategy for the treatment of HIV-associated KS. By blocking CTLA-4 signaling, it may be possible to enhance antitumor immune responses, restore T cell function, and inhibit KS tumor growth. Preclinical studies using animal models of KS have shown promising results with CTLA-4 blockade, highlighting its potential as a novel immunotherapy approach for this malignancy.⁶¹⁻⁹⁰

Preclinical and Clinical Evidence of CTLA-4 Blockade in KS

Preclinical studies investigating the efficacy of CTLA-4 blockade in Kaposi sarcoma (KS) have provided valuable insights into its potential as a therapeutic strategy for this malignancy. Animal models of KS, including murine models infected with murine gammaherpesvirus 68 (MHV-68), have demonstrated the ability of CTLA-4 blockade to enhance antitumor immune responses and inhibit tumor growth. In these preclinical studies, treatment with CTLA-4-blocking antibodies resulted in reduced tumor burden, increased infiltration of effector T cells into KS lesions, and improved survival outcomes, indicating the therapeutic potential of CTLA-4 blockade in KS. Furthermore, early clinical evidence supporting the use of CTLA-4 blockade in KS has emerged from case reports and small pilot studies. In one case report, a patient with HIV-associated KS who failed multiple lines of conventional therapy experienced significant regression of cutaneous KS lesions following treatment with ipilimumab, a monoclonal antibody targeting CTLA-4. Similarly, a small pilot study evaluating the efficacy of ipilimumab in patients with HIV-associated KS demonstrated partial responses and disease stabilization in a subset of patients, suggesting potential clinical benefit. In addition to ipilimumab, tremelimumab, another CTLA-4-blocking antibody, has shown promise in early clinical trials for the treatment of KS. In a phase I/II study involving patients with advanced solid tumors, including KS, tremelimumab monotherapy resulted in disease stabilization and prolonged survival in some patients, with manageable toxicities. These findings support further investigation of CTLA-4 blockade as a therapeutic approach for HIV-associated KS in larger prospective clinical trials. However, challenges remain in the clinical development of CTLA-4 blockade for KS, including the identification of predictive biomarkers to select patients likely to benefit from therapy and the optimization of treatment regimens to minimize toxicities. Moreover, the impact of CTLA-4 blockade on HIV-specific immune responses and viral control needs to be carefully evaluated, given the immunomodulatory effects of this therapy in the context of HIV infection.⁹¹⁻¹²⁰

Challenges and Future Directions

Despite promising preclinical and early clinical evidence supporting CTLA-4 blockade as a potential therapeutic strategy for Kaposi sarcoma (KS) in HIV-infected individuals, several challenges and future directions need to be addressed to optimize the clinical translation of this approach. One of the major challenges in the development of CTLA-4 blockade for KS is the lack of validated biomarkers to predict treatment response and identify patients most likely to benefit from therapy. Future research efforts should focus on identifying biomarkers, such as tumor-infiltrating lymphocyte profiles, immune cell subsets, and molecular signatures within the tumor

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microenvironment, that can reliably predict response to CTLA-4 blockade and guide patient selection for treatment. Optimization of treatment regimens, including dosing schedules, combination therapies, and treatment duration, is essential to maximize the therapeutic efficacy of CTLA-4 blockade while minimizing potential toxicities. Clinical trials evaluating different dosing regimens, combination therapies with other immune checkpoint inhibitors or targeted agents, and treatment duration are needed to determine the optimal treatment approach for HIV-associated KS.¹²¹⁻¹³⁰

Given the immunomodulatory effects of CTLA-4 blockade on T cell function and immune responses, it is crucial to carefully evaluate its impact on HIV-specific immune responses and viral control in individuals living with HIV/AIDS. Longitudinal studies assessing changes in HIV viral load, CD4⁺ T cell counts, and immune activation markers following CTLA-4 blockade are necessary to ensure that treatment does not compromise HIV control or lead to viral rebound. CTLA-4 blockade is associated with immune-related adverse events (irAEs) that can affect multiple organ systems, including the skin, gastrointestinal tract, liver, and endocrine glands. Effective management of irAEs requires close monitoring, early recognition, and appropriate intervention with immunosuppressive agents, such as corticosteroids or tumor necrosis factor- α inhibitors. Future research should focus on optimizing strategies for the prevention, early detection, and management of CTLA-4 blockade-related toxicities in individuals with HIV-associated KS. Given the complex immune dysregulation and tumor microenvironment in HIV-associated KS, combination therapies targeting multiple immune checkpoints, cytokines, and signaling pathways may offer synergistic therapeutic effects. Clinical trials evaluating combination therapies with CTLA-4 blockade and other immunotherapeutic agents, such as programmed cell death protein 1 (PD-1) inhibitors, interleukin-2 (IL-2) agonists, or HHV-8-specific vaccines, are warranted to enhance antitumor immune responses and improve clinical outcomes in individuals with HIV-associated KS.¹³¹⁻¹⁶⁷

Conclusion

CTLA-4 blockade holds significant promise as a novel immunotherapy approach for the treatment of Kaposi sarcoma (KS) in individuals living with HIV/AIDS. Preclinical studies have demonstrated the ability of CTLA-4 blockade to enhance antitumor immune responses, inhibit tumor growth, and improve survival outcomes in animal models of KS. Early clinical evidence from case reports and pilot studies suggests potential clinical benefit of CTLA-4 blockade in HIV-associated KS, with some patients experiencing tumor regression and disease stabilization.

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