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Opinion article

Impact of Immune Dysfunction on Infection Susceptibility in Leukemia Patients

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DESCRIPTION

The human immune system plays a critical role in maintaining homeostasis and defending the body against infections, malignancies, and other pathological insults. Leukemia encompasses a spectrum of disorders including Acute Myeloid Leukemia (AML), Acute Lymphoblastic Leukemia (ALL), Chronic Myeloid Leukemia (CML), and Chronic Lymphocytic Leukemia (CLL). The development of leukemia is driven not only by genetic and epigenetic alterations within hematopoietic progenitor cells but also by profound changes in the immune landscape that influence tumor initiation, progression, and therapeutic response.

The adaptive immune system is particularly affected in leukemia. Exhausted T cells exhibit reduced proliferative capacity, diminished cytokine production, and impaired cytotoxic function. Consequently, the immune system becomes incapable of effectively controlling leukemic growth, allowing disease progression. B cell dysfunction is also observed, leading to impaired humoral immunity, which increases susceptibility to infections and further exacerbates morbidity. These adaptive immune defects demonstrate that leukemia is not merely a disorder of uncontrolled cell proliferation but also a disease marked by systemic immunological compromise.

Innate immune components are similarly dysregulated in leukemia. NK cells, which are critical for early recognition of malignant cells, often exhibit reduced cytotoxicity and impaired cytokine secretion. Monocytes and dendritic cells may show defects in antigen presentation, limiting the activation of adaptive immunity. Neutrophil function can be compromised, leading to defective microbial clearance and contributing to the high risk of infection observed in leukemic patients. Collectively, these innate immune deficiencies create a permissive environment for both leukemic progression and opportunistic infections, compounding clinical challenges.

Genetic and epigenetic alterations intrinsic to leukemic cells further reinforce immune evasion. Mutations in genes regulating the Major Histocompatibility Complex (MHC) can reduce antigen presentation, preventing recognition by CTLs. Epigenetic modifications, such as DNA methylation or histone acetylation, may silence genes involved in immune signaling pathways, further dampening immune surveillance. Moreover, leukemic cells can modulate the expression of immune checkpoint molecules, providing additional mechanisms to escape immune-mediated destruction. This interplay between intrinsic leukemic alterations and immune suppression underscores the complexity of leukemia pathogenesis and highlights the immune system as both a target and mediator of disease progression.

Chronic inflammation also contributes to leukemia pathogenesis by promoting immune dysregulation. Persistent inflammatory signals within the bone marrow microenvironment can induce DNA damage, oxidative stress, and aberrant hematopoietic proliferation. Cytokines such as interleukin-6 and tumor necrosis factor-alpha create a pro-survival environment that favors leukemic clone expansion while simultaneously impairing effective immune responses. Inflammatory signaling can also enhance the recruitment and activation of immunosuppressive cell populations, including Tregs and MDSCs, reinforcing the cycle of immune dysfunction and malignant progression. Thus, immune dysregulation in leukemia is both a cause and a consequence of altered inflammatory states.

The clinical implications of immune dysfunction in leukemia are profound. Immune deficits not only facilitate disease progression but also complicate therapeutic management. Standard treatments, including chemotherapy and radiation, further impair immune function, leading to prolonged cytopenias and heightened infection risk. This immunocompromised state contributes significantly to morbidity and mortality, even when leukemic burden is reduced. Understanding the mechanisms of immune dysfunction has therefore become critical in developing adjunctive therapies aimed at restoring immune competence or harnessing the immune system to target malignant cells.

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