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Commentary

Oncolytic Viruses: A Novel Approach in the Cancer Eradication Effort

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DESCRIPTION

Oncolytic Viral Therapy (OVT) represents an innovative and evolving approach to cancer treatment, utilizing the natural ability of viruses to infect and kill cancer cells while sparing normal cells. Traditionally, cancer therapies have relied on surgery, chemotherapy, and radiation, each with varying degrees of success but significant side effects. With the rise of immunotherapy, the role of Oncolytic Viruses (OVs) in cancer treatment has gained prominence, offering a favorable for both direct tumor destruction and immune system activation.

OVT capitalizes on genetically engineered or naturally occurring viruses that preferentially infect cancer cells. These viruses are either modified to enhance their selectivity for tumor cells or are chosen based on their inherent ability to infect and lyse (break down) malignant cells. In addition to directly killing cancer cells, OVs can stimulate a systemic anti-tumor immune response, thereby helping the body recognize and attack metastatic tumors.

The combination of direct tumor lysis and immune system activation has resulted in the development of several oncolytic viruses, which are currently being tested in clinical trials. One of the most successful examples is the approval of Talimogene laherparepvec (T-VEC), a genetically engineered Herpes Simplex Virus (HSV) for the treatment of advanced melanoma. The encouraging results from this and other trials highlight the potential of OVT as a revolutionary cancer treatment modality.

History of OVT

The concept of using viruses to treat cancer dates back to the early 20th century, when it was first observed that cancer patients with viral infections sometimes experienced temporary tumor regression. However, it was not until the late 1990s and early 2000s, with advancements in molecular biology and genetic engineering, that OVT began to gain significant attention. The ability to genetically modify viruses to enhance their tumor selectivity and safety has been a turning point in the development of OVs as therapeutic agents.

The first oncolytic virus to receive regulatory approval was Oncorine (H101) in China in 2005. It is an adenovirus modified to replicate in and kill tumor cells with defective p53 pathways, a common mutation in cancer cells. In the West, the approval of T-VEC in 2015 by the U.S. Food and Drug Administration USFDA marked a key moment in the clinical application of OVT. Since then, several other OVs have been developed and tested in clinical trials for a variety of cancers, including breast, colorectal, pancreatic, and lung cancers.

OVT works through two primary mechanisms

Direct oncolysis oncolytic viruses selectively infect and replicate within cancer cells. Once inside the cell, the virus hijacks the host's cellular machinery to replicate itself, ultimately causing the cell to burst (lyse) and release new viral particles. This process not only destroys the infected tumor cells but also releases viral antigens and tumor-associated antigens into the tumor microenvironment, which can trigger an immune response.

Defective antiviral responses in cancer cells: Many cancer cells have defects in their antiviral defense mechanisms, such as mutations in the interferon signaling pathways, making them more susceptible to viral infection.

Abnormal tumor vasculature: The disorganized and leaky blood vessels in tumors can facilitate viral entry and spread within the tumor mass.

Genetic engineering: OVs can be genetically modified to enhance their specificity for cancer cells by inserting tumor-specific promoters or deleting viral genes necessary for replication in normal cells.

Immune System Activation: In addition to direct oncolysis, OVs can stimulate the body's immune system to recognize and attack cancer cells. The lysis of tumor cells by OVs releases tumor-associated antigens, Damage-Associated Molecular Patterns (DAMPs), and viral antigens into the tumor microenvironment. These signals recruit and activate immune cells, including dendritic cells, macrophages, and T-cells, which process the antigens and initiate a broader systemic anti-tumor immune response.

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Furthermore, some OVs can be engineered to express immunostimulatory molecules, such as Granulocyte-Macrophage Colony-Stimulating Factor (GM-CSF), to enhance immune cell recruitment and activation within the tumor. This ability to stimulate both local and systemic immune responses makes OVT an attractive strategy, particularly for treating metastatic and immune-resistant cancers.