Lung Cancer and Oncolytic Virotherapy

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Abstract

Lung cancer is one of the most serious threats to human health worldwide, and the covid-19 virus has become people's worst dread since the coronavirus pandemic. There are numerous similarities between cancer cells and viruses, the most significant of which being that both are our adversaries. Oncolytic virotherapy refers to the strategy of using viruses to fight cancer cells. When immunotherapy in the form of immune checkpoint inhibitors has made significant advances in the clinical practice of lung cancer, the induction of antitumor immunity from immune cells has gradually become a rapidly developing and promising cancer therapy strategy. Oncolytic virotherapy is based on the same mechanisms that selectively kill tumor cells and induce systemic anti-tumor immunity, but it is still a long way from becoming a standard treatment for lung cancer. This article provides an in-depth review of the most recent advances in oncolytic virotherapy for lung cancer, including the specific mechanism of oncolytic virus therapy and the main types of oncolytic viruses, as well as the combination of oncolytic virotherapy and existing standard treatments. Its goal is to provide fresh perspectives and ideas on oncolytic virotherapy for lung cancer.

Keywords: Tumor microenvironment •Immunotherapy • Immune checkpoint inhibitor • Non-small cell lung cancer

Introduction

Lung cancer is one of the malignant tumors with the highest morbidity and mortality rates; however, a small number of patients with advanced lung cancer have achieved exceptional therapeutic results from immune checkpoint inhibitors, which are represented by Immunotherapy (ICIs). Another type of cancer immunotherapy with broad anticancer effectiveness is Oncolytic Virotherapy (OV). The idea of employing an oncolytic virus to treat cancer has now been proven in multiple recent clinical trials, and a real therapy for melanoma has been approved by the FDA. A systematic review and meta-analysis of the efficacy and safety of OV in lung cancer found that patients receiving oncolytic adenovirus H101 monotherapy or in combination with chemotherapy had a significantly higher objective response rate than patients receiving only chemotherapy [1-5].

OV is becoming increasingly useful in cancer therapy because it combines direct oncolysis with antitumor immune activation. Oncolytic viruses are designed to infect and replicate preferentially in tumors, promoting not only immunogenic cell death but also increasing the number of immune mediators entering the tumor microenvironment, disrupting intratumoral immunosuppression and inducing systemic antitumor immunity in the host. Enadenotucirev (previously known as ColoAd1), a tumor-selective chimeric adenovirus, was found to stimula--telocal high antitumor immune Responses such as CD8+ T cell infiltration in resectable Non-Small Cell Lung Cancer (NSCLC).

Furthermore, vaccinating high-risk populations with an oncolytic adenovirus or vaccinia Virus-Infected Reprogrammed Somatic-Derived Tumor Cell Vaccine (VIReST) regimen can prevent tumor progression and initiate long-term anti-tumor immune response monitoring, which can be used in the treatment and prevention of lung cancer. Furthermore, computers were able to find ideal vaccine candidates for components of an oncogenic virus that causes lung cancer, and these epitopes have a high therapeutic promise as lung cancer vaccines. Preclinical and clinical trials of OV for lung cancer have shown numerous breakthroughs.

Tumor-associated stroma limits oncolytic virus efficacy by forming barriers that prevent efficient virus penetration and spread; more importantly, their viral therapeutic efficacy is severely hampered by limited viral spread and negative immune regulation in the tumor microenvironment. One of the drawbacks of intravascular oncolytic virus delivery is that systemic administration will be hampered by antiviral immunity. Adenoviral vector Ad5-3M was obtained in some studies by targeting functional sites in the viral capsid. When oncolytic virus was administered to mice with lung cancer, researchers discovered that the virus not only replicated in tumor cells, but also resisted antiviral immunity.

Blood Outgrowth Endothelial Cells (BOEC) can be used to deliver the interferon—expressing oncolytic vesicular stomatitis virus VSV-IFN vector in a preclinical model of NSCLC, with VSV - IFN-infected human BOECs showing excellent antitumor activity and mouse survival in immunodeficient A549 xenograft model mice, and infected BOECs killing NSCLC cells in In addition, microfluidic encapsulation of oncolytic adenovirus displayed good in vivo anticancer effect against *A549* lung tumor-bearing mice by suppressing proliferation, increasing oncolysis, and perhaps immunomodulating. Systemic administration of oncolytic adenovirus and paclitaxel encapsulated in Extracellular Vesicle (EV) preparations significantly increased transduction rates and infectious titers in vitro, while effectively inhibiting the growth of human lung cancer in mice. A second important restriction of oncolytic virus therapeutic efficacy is antagonism caused by limited diffusion into solid tumors. *AdUV* was created by repeatedly exposing viral particles of the oncolytic adenovirus wild-type *Ad5 dl309* to C-type UV irradiation, which has been demonstrated to more efficiently lyse cancer cells. Furthermore, the function of oncolytic viruses is dependent on the immune response in the tumor microenvironment, and in the treatment of lung cancer, the combination therapy of interleukin 10 (IL-10) and oncolytic adenovirus *Ad-hTERT* improved antitumor efficacy.

Systemic treatment of the IL-12-expressing *NV1042* virus is also more successful than its non-cytokine parent, *NV1023*, in treating metastatic lung cancers. In a variety of tumor types, the use of released or membrane-bound IL-23 vaccinia virus causes anticancer effects.

Effective immunotherapy necessitates simultaneous targeting of cancer cells and immunosuppressive stromal cells, and the third major limitation of viral therapy for disseminated cancer is poor tumor targeting of oncolytic adenoviruses after systemic administration. Some research suggests that tumor-infiltrating T cells can be more efficiently activated and redirected by oncolytic adenoviruses equipped with Bispecific T Cell Engager (BiTE) antibodies, and that arming oncolytic adenoviruses with BiTEs could overcome OV's "targeted limitation." An oncolytic group B adenovirus modified with BiTEs could target tumor cells as well as immunosuppressive stromal cells. This BiTE binds FAP on Cancer-Associated Fibroblasts (CAFs) and CD3 on T cells, leading in effective T cell activation and fibroblast killing. Because FAP is overexpressed in CAFs, oncolytic adenovirus

OAd-FBiTE armed with FAP-targeted bispecific T cells can retarget infiltrating lymphocytes to CAFs, increasing viral dissemination and T cell-mediated cytotoxicity targeting tumor stroma. Another oncolytic herpesvirus that expresses PD-L1 BiTE, which causes a pro-inflammatory response and destroys cells that drive tumor development, minimizes systemic toxicity [6].

T cell recruitment and activation in the tumor microenvironment were greatly boosted by a novel recombinant oncolytic virus VV, VV—TIGIT, encoding a completely monoclonal antibody against T cell immunoglobulin and ITIM domain (TIGIT). Another modified oncolytic vaccinia virus, VV-scFv-TIGIT, elicits effective antitumor immunity by encoding a Single-Chain Variable Fragment (scFv) targeting T-cell immunoglobulin and the ITIM domain. To improve anticancer efficacy, oncolytic viruses producing antibodies against immune checkpoint domains successfully combine the benefits of OV with intratumoral production of ICIs [7,8].

Conclusion

Until today, it has become an unmistakable fact that we cannot destroy all tumor cells, just as we cannot eliminate coronavirus, and that learning to live with them is the best option. OV is one of the most appealing current experimental treatments for human diseases, with a great antitumor effect on malignant tumors. However, the FDA has only approved one OV so far: T-Vec, a modified form of herpes simplex virus type 1 (HSV-1). As a result, it deserves our undivided attention to continue developing novel oncolytic viruses, conducting basic research on the specific immune modulation mechanisms that enhance oncolytic virus replication and tumor-killing rates, and developing oncolytic combination therapies that can synergize with other existing standard treatments for lung cancer. More importantly, developing a vaccination system to prevent tumor progression and to initiate long-term anti-tumor immune monitoring is critical for lung cancer treatment and prevention.

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