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A Brief Report on Effect of Human Cytomegalovirus Infection in Ovarian Cancer

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Introduction

One of the most common, aggressive, and potentially fatal gynecological diseases affecting women is ovarian cancer (OC). Tumors of the ovary can be caused by germ cells, stromal cells, or epithelial cells. In developed nations, epithelial OC accounts for more than 90% of malignant OC. The 5-year survival rate for approximately 75% of all cases of OC is less than 30%, and the disease is diagnosed at an advanced stage (stage 3–4). Primary debulking surgery, followed by platinum-based chemotherapy with paclitaxel and carboplatin, is the current standard of care for women with OC. However, recurrences occur in almost 66% of patients within two years of diagnosis, with patients diagnosed at a later stage accounting for the majority of these recurrences [1].

Description

Human cytomegalovirus (HCMV), also known as human herpes virus 5 (HHV-5), affects approximately 83 percent of the world's population, with close to 100 percent of that number living in developing nations. HCMV establishes a lifelong chronic latency in humans following primary infection, primarily in the bone marrow's cluster of differentiation (CD)34+ hematopoietic progenitor cell population. In immunocompetent individuals, latent infection is typically asymptomatic, but symptomatic reactivation can occur, particularly in immunocompromised or cancer patients. Latent HCMV reactivation is characterized by high levels of circulating pro-inflammatory cytokines, especially when CD34+ progenitor cells become inflammatory monocytes, infiltrating macrophages, or dendritic cells. These cells then spread the virus to peripheral organs and body tissues, infecting and replicating in a wide variety of cell types.

The host-protective and anti-tumor mechanisms that the human immune system uses to stop ovarian tumors from growing. The immune system's most effective strategy for preventing the development of OC is cell-mediated cytotoxicity, which involves two main types of cells: Natural killer (NK) cells and CD8+ cytotoxic T cells (CTLs). Two steps are used by CTLs to perform their effector mechanisms: Granule-mediated killing is followed by interaction with the T cell receptor (TCR) by MHC class I. A polarization of the CTL that ensures a level of organization between the CTL and the target cell occurs when the TCR recognizes the MHC-I antigen on CTLs. The CTL undergoes morphological changes and releases lytic granules that kill target cells, such as perforin and granzymes (granule enzymes). The polymerization of perforin results in the formation of pores in the target cells, which permit granzymes like granzyme B to enter the target cells. Granzyme B promotes apoptosis by activating caspase, a family of protease enzymes involved in programmed cell death, and by promoting BID, a member of the BH3 domain-containing proapoptotic Bcl2 family. Due to the high presence of Tregs in the tumor microenvironment (TME), which typically inhibit CTL responses, data from previous studies have demonstrated that the developing tumor has a significant impact on the immune system of OC patients. Recent research has demonstrated a strong correlation between OC recurrence and the immune status of the TME, including the presence of pro-inflammatory cytokines and Tregs and the absence of tumor-infiltrating CD8+ T cells. Conversely, the significance of CTL-mediated immune responses in OC is highlighted by the fact that the presence of tumor-infiltrating CD8+ T cells and a high CD8+ T cell/Treg ratio is linked to significantly improved survival outcomes.

Additionally, an immune system that reacts too quickly can damage tissue if it does not resolve. The immune system uses immune checkpoint inhibitory pathways, which are necessary for ensuring self-tolerance and regulating the extent and magnitude of CTL and NK cell effector responses, to reduce such damage. Surface inhibitory receptors like cytotoxic T lymphocyte antigen-4 (CTLA-4) and programmed cell death protein 1 (PD-1) are involved in these inhibitory pathways. CD279). Under normal circumstances, they are typically expressed only briefly on activated T cells, B cells, macrophages, dendritic cells, and Tregs; however, prolonged or increased expression is a sign of T cell exhaustion [2-5].

Conclusion

There is currently a significant amount of research interest in personalized medicine, not only for OC but also for a wide range of other cancers. Through impairment of CTL and NK cell responses, which promotes their survival or progression, HCMV and OC share similar disease mechanisms. Numerous cell types can be infected by HCMV, as previous research has shown; induces an environment that encourages inflammation and tumor growth. In addition, active HCMV infection results in the formation of an immunosuppressive TME that suppresses immune responses specific to the tumor. The use of an anti-HCMV strategy in the treatment of OC patients who are infected with HCMV has never been the subject of a clinical study. However, anti-HCMV targeted T cell therapy has produced some positive treatment outcomes in GBM patients, particularly in recurrent GBM patients. As a result, it is reasonable to hypothesize that personalized anti-HCMV treatment may contribute to an improvement in the survival rates of OC patients, particularly those with an active HCMV infection in their TME. Patients with HCMV-positive ovarian tumors need additional research to determine whether the use of anti-HCMV therapy in conjunction with current, well-established first-line and second-line therapies is effective in increasing survival rates.

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Conflict of Interest

The authors declare that there is no conflict of interest associated with this manuscript

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