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Emerging Immune Modulators in Cancer: Anthelmintic Drugs

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Introduction

In spite of late advances in therapy draws near, disease is as yet one of the main sources of death around the world. Restoring tumour immune surveillance is a valid strategy for overcoming the acquired resistance and cytotoxicity of conventional oncology therapies and immunotherapeutic drugs like immune checkpoint inhibitors and immunogenic cell death inducers. It has also made significant progress in the treatment of a number of cancers and improved the clinical management of advanced diseases. Unfortunately, only a small percentage of patients clinically respond to and benefit from cancer immunotherapy due to tumour-intrinsic and/or tumour-extrinsic mechanisms for evading immune surveillance. Evidence from studies of drug repositioning, or the strategy of finding new uses for approved or investigational drugs that go beyond the original medical indication, has shown that some anthelmintic drugs have important immunomodulatory effects on specific subsets of immune cells and related pathways in addition to their antineoplastic effects. The current understanding of how anthelmintic drugs affect host immunity and how they might be used in cancer immunotherapy is presented in this review.

Description

Cancer is a significant obstacle to increasing life expectancy worldwide and one of the leading causes of death. An estimated 19.3 million new cases of cancer and nearly 10.0 million deaths from cancer were recorded across all countries. With an estimated 2.3 million new cases, female breast cancer has overtaken lung cancer as the most common cancer to be diagnosed, accounting for 11.7% of all new cases. Other common cancers include lung (11.4%), colorectal (10.0%), prostate (7.3%) and stomach (5.6%). With an estimated 1.8 million deaths, lung cancer continues to be the leading cause of cancer death, followed by colorectal (9.4%), liver (8.3%), stomach (7.7%) and breast (6.9%). The burden of cancer-related morbidity and mortality worldwide is generally increasing rapidly. This reflects both maturing and development of the populace, as well as changes in the pervasiveness and conveyance of the primary gamble factors for malignant growth, a few of which are related with financial turn of events. Surgical removal is currently the primary focus of solid cancer therapy, despite the fact that this option is typically only available in the early stages of cancer development. Chemotherapy is the treatment of choice when metastases are discovered in the later stages. Sadly, a significant number of patients may develop resistance to cancer medications at the outset or during treatment, eventually leading to therapy failure. Researchers are forced to look for novel chemicals as a result of these issues, which necessitate additional therapeutic options [1,2].

Nonetheless, the improvement of new therapeutics has become progressively challenging for drug organizations. In point of fact, target identification and validation, evaluation of compound efficacy and pharmacology and evaluation of toxicology, specificity and potential drug interactions are all necessary components of traditional drug discovery. As a result, putting a new drug on the market requires a lot of time and money and can take anywhere from 10 to 15 years to complete. An overall failure rate of more than 96% in drug development (with a failure rate of 90% during clinical development) and a decreasing number of new therapeutics approved by drug regulatory authorities demonstrate the scope of these obstacles.

Because of the levels of proinflammatory cytokines and T-cell infiltration, the tumor microenvironment can be simply described as "cold" (non-T-cell-inflamed) or "hot" (T-cell-inflamed). T-cell infiltration and molecular evidence of immune activation distinguish these so-called "hot" tumors from "cold" tumors, which are marked by T-cell absence or exclusion. Immunotherapeutic drugs that target programmed death protein 1 (PD-1) and its ligand, programmed death-ligand 1 (PD-L1), which mediate co-inhibitory signals to T-cell activation and reduce the host immune response to tumor cells, generally respond better to hot tumors [3].

In this regard, it is essential to emphasize that the cytostatic and pro-apoptotic properties mentioned earlier are not the only potential antitumor effects of various anthelmintic drugs, as several studies highlighted their ability to alter host immunity. These compounds, which are involved in the immune response to cancer, can have an impact on the activity of various immune cells and transcription factors, such as STAT3 (signal transducer and activator of transcription 3) and NF-B (nuclear factor kappa light chain enhancer of activated B cells). In addition, some anthelmintics have been shown to work in conjunction with PD-1/PD-L1 blockade immunotherapy or inducing immunogenic cell death (ICD), a particular type of apoptosis linked to endoplasmic reticulum (ER) stress and the release of damage-associated molecular patterns (DAMPs).

In oncology, restoring tumor immune surveillance is a viable strategy for overcoming conventional therapy cytotoxicity and acquired resistance. Immunotherapeutic drugs, such as immunogenic cell death inducers and immune checkpoint inhibitors, have made significant progress in the treatment of a number of cancers and improved the clinical management of advanced disease [4].

Due to tumor-intrinsic and/or tumor-extrinsic mechanisms for evading immune surveillance, only a small percentage of patients clinically respond to and benefit from the aforementioned treatments. In addition, in some patient populations, some immunotherapy-related side effects, such as toxicity, unwanted immune-mediated reactions, hyper- and pseudo-progression of the disease, may jeopardize survival and quality of life. In recent years, experimental evidence has shown that some anthelmintic drugs play an unexpected role in cancer treatment by altering the immune response of the host [5].

Conclusion

By inhibiting the infiltration of immunosuppressive cell subsets (i.e., MDSCs) into the tumor niche and by boosting antigen presentation by dendritic cells and/or Th1-mediated/cytotoxic immune responses, some anthelmintic drugs, for instance, have been shown to subvert an immunosuppressive tumor microenvironment. As a result of all of these findings, significant efforts were made to test the most promising compounds in conjunction with immune checkpoint inhibitors or ICD inducers in order to boost the success of immunotherapy. This review's findings suggest that some anthelmintic drugs may have a promising future as adjuvant agents in cancer immunotherapy. However, they have not been adequately or properly evaluated in clinical trials and in order to increase the likelihood of success in clinical practice, some disadvantages must be addressed and overcome.

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