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Maladaptive Immunity and Metastasizing Cancer

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Abstract

The ability of innate immunity to inhibit metastatic cells is limited, based on Stage IV cancer survival rates. The dysregulation of the immune system through acquired immunity may result in pathological conditions that alter metastatic cells. Immunoglobulin-E (IgE) antibodies developed by the humoral immune system are a significant contributor to maladaptive immunity. Hypersensitivities are maladaptive immune reactions against harmless allergens. Forced allergen-specific immune responses may provide immediate-type allergies that affect the incidence and prevalence of endogenous proteins essential for metastasizing cells. Furthermore, allergies may shift the body's resource allocation away from metastasizing cells to IgE-primed effector-cell proliferation. Therefore, research efforts need to explore if hyper-allergenic skin creams can be used to starve-out metastatic cells, wherever they are in the body, to determine if maladaptive immunotherapy is a viable treatment for Stage IV cancer.

Keywords: ***Maladaptive immunity; Immunoglobulin-E (IgE) antibodies; Stage IV cancer***

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Introduction

In a communication from the Centers for Disease Control and Prevention (CDC) titled, Deaths: Final Data for 2017, statistics showed that malignant neoplasms (cancer) were the second leading cause of death [1]. Most deaths (about 90%) associated with cancer are due to the metastasis of the primary tumor cells to sites distant from the initial or primary tumor. Metastasis is the process by which cancer cells migrate throughout the body. Migrating cancer cells can die for a variety of reasons. Cells typically live tightly connected to their neighbors' detachment from the surface of other cells can lead to cell death. Furthermore, cancer cells are often quite large in comparison to the cells that typically live in the lymphatic system or blood system. When they travel through the vessels, they can get damaged or stuck, leading to cell death. Finally, cancer cells can be recognized and destroyed by cells of the immune system [2].

What role does immunity play in metastasis? In a review article from the Journal for Immunotherapy of Cancer, the authors write, "As metastases are considered to be secondary tumors derived from the primary tumor after its establishment, concomitant immunity may be involved in controlling the occurrence of metastasis. Because the immune system can both promote and inhibit metastasis, it is of great importance for the clinic to understand which mediators are involved and how they impart their effects, to identify new targets to prevent metastatic disease [3]."

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Spontaneous healing of cancer continues to be a controversial subject. In an article from the Journal of Natural Science, Biology and Medicine titled, Immunity over inability: The spontaneous regression of cancer, the author writes, "It is interesting to note that the current primary cancer management procedures neither harness the benefits of patients' own immune system nor stimulate it to achieve tumor regression but actively suppress it; thus it does not run parallel to body's own defensive mechanisms but opposes its natural role. An ideal cancer management would involve the stimulation of the immune system, its complex effective and reproducible *in vivo* mechanisms that fight cancer [4]."

Cancer immunotherapy is a form of treatment that uses the power of the body's innate and humoral immune system to inhibit and eliminate cancer. In an opinion article from STAT titled, few people actually benefit from 'breakthrough' immunotherapy the authors write, "When immunotherapy works, the result is terrific, even life-changing. Today, though, only a tiny minority of patients expected to die from cancer will benefit from immunotherapy [5]."

Most recently, the dysregulation of the immune system (i.e., Natural Allergy- Oncology) has been hypothesized to inhibit tumor growth and metastasizing. Repeated exposure to allergens induces the human body to form B-cells, IgE antibodies, and IgE-primed effector cells, which bind to the allergen to begin the process of elimination and removal. Suppression of metastatic cancer is suspected with IgE antibodies in that they are exceptionally biologically active despite being present in relatively low concentrations in the bloodstream, i.e., approximately one-thousandth of a percent. B-cells produce IgE antibodies that bind to high-affinity receptors on the surface of effector cells (e.g., mast cells, basophils, and eosinophils) to provide IgE-primed effector cells. The IgE-primed effector cells bind to allergens and endogenous proteins, having structure homology to expedite their removal. In a research article from the Journal of Allergy titled, Allergo-Oncology: The role of IgE-mediated allergy in cancer, the authors write, "IgE antibodies may not only act in natural tumor surveillance but could also be exploited for tumor control in active and passive immunotherapy settings [6]." Furthermore, effector cells that support humoral immunity have been shown to affect early and late-stage cancer [7]. The emerging picture from most of the currently available epidemiological data indicates that atopic disease is associated with a reduced risk for cancer [8].

Discussion

An immediate type hypersensitivity (allergy) is a maladaptive immune response towards a harmless foreign substance (i.e., allergen) that occurs within minutes of exposure. IgE-primed effector cells can be used to induce a cross-react immune response, through molecular mimicry, with endogenous proteins that have structure homology based on conformational and linear epitopes [9,10] A research article proposes that a forced allergen-specific immune response may provide an immediate-type hypersensitivity that affects the incidence and prevalence of endogenous proteins essential for metastasizing cells [11].

Patients with terminal stage IV cancer often use conventional medical treatments (e.g., chemotherapy, radiotherapy, targeted cancer drugs, and a high dose of steroids) to prolong survival and ease symptoms, although, these treatments temporarily weaken the immune system and fail to cure the disease. Such treatments compromised the immune systems based on a drop in the number of white blood cells made in the bone marrow [12]. Discovering new ways of activating a compromised immune system may improve outcomes. Repeated and extended exposure to exogenous antigens may increase a maladaptive pattern of immunological reactivity to increase cancer survival rates [13].

The skin contains elements of humoral immunity (i.e., adaptive immunity), allowing it to be hypersensitive to allergens. Atopic dermatitis is associated with the presence of IgE antibodies, while allergic contact dermatitis is a T-cell-mediated delayed-type hypersensitivity reaction. The 'Hapten Atopy Hypothesis' postulates that normally 'harmless' environmental allergens delivered via skin will initially stimulate a Th1 immune response, but repeated and prolonged exposure to such allergens in genetically predisposed individuals will likely shift the response from Th1 to Th2 leading to antibody class switch in B-cells producing IgE antibodies which then tightly bind to peripheral mast cells and basophils (i.e. effector cells). Subsequent exposure to the allergens causes cross-linking of the allergen and IgE-primed effector cells to form a complex, inducing degranulation of the complex with the release of histamine and inflammatory agents [14].

Topical hyper-allergenic compositions (e.g., skin creams) have been proposed to affect the incidence of specific maladaptive immune responses during stage IV cancer. It is intended to halt the progression of metastatic cancer using natural and recombinant allergens utilizing a skin cream that can be self-applied [15]. If a cancer patient has a compromised immune system, can the use of a hyper-allergenic skin cream induce atopy through epigenetics? In a research article from Allergy, Asthma & Clinical Immunology titled, Genetic and epigenetic studies of atopic dermatitis (AD), the authors concluded, "The results suggest that two major biologic pathways are responsible for AD etiology: Skin epithelial function and innate/adaptive immune responses. The dysfunctional epidermal barrier and immune responses reciprocally affect each other, and thereby drive development of AD [16]."

Hormone therapy may be a useful supplementary treatment during maladaptive immunotherapy. Sex hormones affect the immune system by increasing the number of circulating immune cells. In a review article from The Scientific World Journal titled, Sex hormones and immune dimorphism, the authors write, "Estrogen and testosterone are major regulators of the immune system. Sex hormones of the reproductive system are one of the major factors that regulate the immune system due to the presence of hormone receptors on immune cells. The interaction of sex hormones and immune cells through the receptors on these cells affects the release of cytokines, which determines the proliferation, differentiation, and maturation of different types of immunocytes and as a result, the outcome of inflammatory or autoimmune diseases." The authors suggest that the modulation of cytokine production through hormones may help in the future to design potential targets for therapy in sex hormones related to inflammatory or autoimmune diseases. [17]

Conclusion

Skin creams that induce allergies are a new approach to cancer immunotherapy. Allergies are a relative contraindication in that the risk of complications from many allergies does not preclude the condition of Stage IV cancer. Although maladaptive immunotherapy is a non-conventional approach, research efforts need to explore if forced hypersensitivity to specific allergens can cause metastatic cells to become benign.

Conflict of Interests

The author is co-founder of Alleam, LLC. Minnesota, USA.

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