Review on Cancer and the Immune System

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Abstract

Cancer is a term for a large group of diseases caused by abnormal cells divide rapidly and spread to other tissue and organs. Under normal circumstances, so many cells multiply as long as the body is in need of them for the proper function daily. Healthy cells have a particular life cycle, reproducing and dying off in a way that is determined by the kind of cell. New cells replace old or damaged cells when they die. On the other hand, abnormal growth of cell or cancer disrupts this normal function. Cancer is one of the leading causes of death worldwide. It can migrate through the blood vessels or lymphatic system to different areas throughout the body. With regard to cancer diagnosis: one method is detection of a compromised immunological response of the patient toward his own tumor cells. And the second method is to use antisera, with which immunization of animals with human tumor extracts, for the detection of substances released into the blood by the tumor cells.

Keywords

Cancer; Tumor Cells; Lymphatic System; Natural Killer Cells
Introduction

A major function of the immune system is the discrimination of self from nonself. As multicellular organisms evolved, it became imperative that they recognize foreign invaders, such as viruses, bacteria and fungi and that they recognize damaged or dysregulated self, such as tumor cells. Cancer can be defined as a disease in which a group of abnormal cells grow uncontrollably by disregarding the normal rules of cell division [1]. Normal cells are constantly subject to signals that dictate whether the cell should divide, differentiate into another cell or die. Cancer cells develop a degree of autonomy from these signals, resulting in uncontrolled growth and proliferation [2]. In males the dominant cancers include lung cancer, prostate cancer, colorectal cancer and stomach cancer. As per study result of Marusyk, et al., revealed that, the most common types are breast cancer, colorectal cancer, lung cancer and cervical cancer in women [3].

Now that a vast amount of theoretical, experimental and clinical data have been accumulated in the field of molecular and cellular oncology about the multifaceted diversity of mechanisms and factors of carcinogenesis and anti-carcinogenesis, it seems reasonable to revive an interest and revise some of the concepts of the tumor-host relationship [4].

Local symptoms may occur due to the mass of the tumor or its ulceration [5]. For example, mass effects from lung cancer can block the bronchus resulting in cough or pneumonia; esophageal cancer can cause narrowing of the esophagus, making it difficult or painful to swallow; and colorectal cancer may lead to narrowing or blockages in the bowel, affecting bowel habits.

Tumor development can be controlled by cytotoxic innate and adaptive immune cells [6]. In addition, Bhardwaj, described that chronic inflammation is a character of cancer, with at least 25% of cancers associated with it and possible factors that contribute to cancer include microbial infections, autoimmunity and immune deregulation. Furthermore, Human Papilloma Viruses (HPVs) induce inflammation and are responsible for 90%-100% of all cervical cancers. Similarly, chronic infection with Helicobacter pylori can elevates the risk for gastric cancer.

In this review, it has been tried to address the recent advances of the contributions of local and systemic environments to cancer progression, the ability of tumors to actively disrupt the host environment and diagnostic and therapeutic approaches that are designed to control cancer.

Tumor and Host Relationship

Tumours are quite different from inflammatory or other swellings because the cells in tumours are abnormal in appearance and other characteristics [7]. It has been given attention to know how immune cells affect tumor fortune in different categories, for example, at an early
neoplastic transformation, in the clinically detected tumors, at metastatic dissemination and at therapeutic intervention [8]. Immunodeficiency can predispose to tumor development [9]. Bhowmick, et al., reveals that the relationship between carcinoma and cell of host [10]. Furthermore, human carcinomas and or tumors can be induced by fibroblasts, myo-fibroblasts, adipocytes, endothelial cells, pericytes and immune cells.

### An Immune Response to Tumors

Suppressor T-cells inhibit the immune response against tumors [11]. Natural Killer (NK) cells mediate innate immunity and contain huge numbers of perforin- and granzyme-rich granules, which empower them to lyse NK-sensitive tumor targets without prior antigen sensitization or clonal expansion. Immunosuppressed transplant recipients have an increased risk of developing certain cancers, including cancers without a known viral aetiology, such as melanoma [11,12].

A likely explanation of how T-cell responses to tumors are initiated is that tumor cells or their antigens are ingested by host APCs, particularly dendritic cells and tumor antigens are processed inside the APCs (Fig. 1). Peptides derived from these antigens are then displayed bound to Class I MHC molecules for recognition by CD8+ T-cells. The APCs express costimulators that provide the signals needed for differentiation of CD8+ T-cells into anti-tumor CTLs [13].

![Figure 1: Immune response to tumors.](image-url)
Almost 90% of cancer-related deaths are due to tumor spreading a process called metastasis [2]. Unstable tumor genomes contain many mutations that generate altered protein products, which have the potential to be recognized as foreign by the host immune system during surveillance [14].

Morishita, et al., described tumor antigen as an antigenic substance produced in tumor cells, i.e., it triggers an immune response in the host [15]. Tumor antigens are useful tumor markers in identifying tumor cells with diagnostic tests and are potential candidates for use in cancer therapy.

**Sufficiency of the Immune System to Eliminate Tumor Growth**

Many molecules and cells of the immune system participate in the recognition and destruction of cancer cells [16]. According to Wang, et al., indicates tumour-specific antibody responses (also known as antibody signatures) can be used to detect cancers such as prostate cancer at early stages [17]. Moreover, antibodies specific for cyclin B1 or the tumour-suppressor protein p53 might be useful for detecting cancer at early stages [18].

**Diagnosis of Cancer**

Considerable importance is attached to diagnosing cancer at an early stage, since this greatly increases the chances of curing the disease. Mass screening techniques have been particularly successful against cervical cancer, where women are encouraged to attend a clinic every three years at which a smear is taken painlessly from the epithelial tissue at the neck of the womb. The cells are examined under the microscope in order to check for a ‘pre-cancerous’ state. In addition, ELISA is mostly used to screen for and to follow the status of prostate cancer and colon and liver cancer. This supplies data that are much more useful to the physicians in treatment than used to be available through ordinary tissue section examination.

**Treatment of Cancer**

Treatment of cancer may be by surgery to remove the tumor, by radiotherapy to destroy the cancerous cells, by chemotherapy with drugs or by combinations of all three.
Future Perspectives

Through a deeper understanding of the complicated relationship between tumors and the immune system, tumor immunology strives to harness the immune system to generate protective antitumor responses in patients [19]. An understanding of the interaction between immune cells, tumour cells and treatment modalities will therefore guide the future combination of immunotherapy with conventional therapy to achieve optimal anti-tumour effects [20,21]. Potential strength of immune surveillance is based on the knowledge that cancer cells express tumour associated antigens that can be recognised by the immune system as foreign elements. Furthermore, it is reasonable to expect more practical screening and diagnostic applications.

Conflict of Interest

Authors declare no conflict of interest.

References