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Related Advances in Tumour Immunity

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1. Preface

Cancer is a heterogeneous group of diseases and the immune tumour microenvironment is an important factor in the pathogenesis of many cancers. Immune patterns mediate the complex relationship between the heterogeneity of immune infiltrating cells, the tumourphenotype and the response to cancer therapy. After recognition by the immune system, tumours may be attacked by the immune system through immunosurveillance processes, with mucosal immunity representing the first line of defence, and cellular and humoral immunity also playing an important role in carcinogenesis and disease progression. The association of the patient's peripheral blood and tumour microenvironment also reveals the link between different types of cancer and immunology.

Breast cancer and immunity

Breast cancer is a heterogeneous disease that is traditionally classified into three phenotypes: ER-positive, HER2-positive and triple-negative. Breast cancer is characterised by a highly inflammatory microenvironment maintained by infiltrating immune cells, cytokines and growth factors. Immune infiltration in breast tumours is also associated with clinical outcomes in terms of therapeutic response modulation; for example, the presence of large numbers of regulatory T cells is associated with poor prognosis in ER-positive and ER-negative breast tumours, where the mechanism is an immunosuppressive environment [1]. A large proportion of natural killer cells and neutrophils, and a smaller proportion of CD8+ and CD4+ T cells, were found in ER-positive breast tumours. Eosinophils and monocytes were associated with a good response after chemotherapy; B lymphocytes were associated with a good prognosis for this phenotype; and activated mast cells were associated with a good prognosis. In addition, tumour-associated macrophages TAM1 and TAM2 and regulatory T cells contribute to a poorer prognosis due to their inflammatory, immunosuppressive and tumour-promoting effects [1]. In ER-negative breast tumours, the main components of immune infiltrating cells are regulatory T cells, TAM 2 and activated mast cells, which are also associated with poor prognosis. In contrast, CD4+ T cells, CD8+ T cells, B lymphocytes and dendritic cells are associated with a better prognosis and may be associated with a good response to neoadjuvant chemotherapy. For HER2-positive breast cancer types, the immune cells in the tumour consist mainly of dendritic cells, mast cells, γδ T lymphocytes, regulatory T cells and neutrophils, all of which contribute to the poor prognosis, disease recurrence and metastasis of this phenotype [2].

Cervical cancer and immunity

The development of cervical cancer is directly linked to HPV infection and, on the other hand, a defective immune system plays an important role in cancer progression [3]. HPV infection triggers a predominantly cell-mediated immune response, with T helper cells involved in the clearance of lesions, and it has been shown that Langerhans cells are increased in women who clear HPV. HPV is divided into low and high risk groups based on its oncogenic potential, with the different groups stimulating similar cellular environments and immune defences but each having different pathological and cellular targets, for example the low risk HPV E7 protein has a lower binding affinity [4].

Glioma and immunity

Immune cells in malignant gliomas can 'make peace' with tumour cells and other microenvironmental components in the tumour microenvironment, creating a microenvironment conducive to glioma cell proliferation; in the glioma microenvironment glioma-associated microglia/ macrophages and regulatory T cells exert immunosuppressive effects, while natural killer cells with anti-tumour effects are inactivated. The glioma-associated microglia/macrophages and regulatory T cells play an immunosuppressive role in the glioma microenvironment, while natural killer cells with anti-tumour effects are inactivated. The suppression of the immune system in malignant gliomas is not only localised to the tumour but also systemic [5]. It is now known that T cells from the deep cervical lymph nodes can enter the brain through the meningeal lymphatics. In the pathological state of malignant glioma, not only are there few T cells left in the deep cervical lymph nodes, but there are also less than 1/3 of the normal T cells in the peripheral circulation, which have been shown to be recalled by the bone marrow of patients with malignant glioma [6]. When T cells in the immune system are completely depleted or recalled, there is no limit to the growth of malignant glioma cells.

2. Prospects

The understanding of the formation of the tumour immunosuppressive microenvironment has led to the development of immune checkpoint-targeted therapy, but the efficacy of this therapy in glioblastoma is not satisfactory, because when immune checkpoint-targeted therapy is administered, tumour-killing T cells are still systematically recruited rather than locally derived from the tumour.

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