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Commentary

Anisotropy of Macrophages in Ovarian Cancer

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DESCRIPTION

One of the most frequent gynaecological cancers is ovarian cancer. Chemotherapy resistance, severe intraperitoneal metastasis, and other factors are responsible for the high mortality rate of ovarian cancer patients. Patients with advanced serous ovarian cancer have a death rate as high as 70%. According to analysis, the tumour microenvironment has a significant impact on the course and prognosis of ovarian cancer. Due to the existence of tumor-infiltrating lymphocytes, it is established if a tumour is "hot" or "cold," and "hot" tumours are those that have lymphocytic infiltration while "cold" tumours are the opposite. Specifically, solid tumours can be classified into four subtypes, referred to as TMIT I to IV, depending on the expressions of PD-L1 and TILs. In a nutshell, "hot" and "cold" indicate whether or not the tumours are immunogenic. Due to the high density of lymphocytes infiltrating "hot" tumour, immunosuppressive checkpoint inhibitors can be used to treat it. Ovarian cancer has a rather high tumour mutational burden, yet it still falls under the category of "cool" tumours. Future study will concentrate on determining how to stimulate the immune system, particularly T cells and TAMs, in "cold" tumours.

Macrophages have a significant role in the microenvironment of tumours. A significant fraction of the tumour stromal cells are Tumour-Associated Macrophages (TAMs), which are macrophages produced by the infiltration of peripheral blood monocytes into solid tumour tissues. The developments in knowledge of macrophages in ovarian cancer, particularly the role of TAMs in ovarian cancer biology and prognosis. This may be a successful new approach for the treatment of ovarian cancer, according to a significant number of fundamental and preclinical investigations that target macrophages.

Macrophage polarization in ovarian cancer

The tremendous adaptability and variability of macrophages allows them to perform a variety of biological tasks in various microenvironments. M1 macrophages, or traditionally activated macrophages, and M2 macrophages, or alternatively activated macrophages, are the two main phenotypes of macrophages and are activated in various ways. Note that there are at least three different subtypes of M2 macrophages, including M2a, which is activated by IL4 and IL13, M2b, which is induced by immune complexes or LPS, and M2c, which is induced by IL10, TGF-, or glucocorticoids. Macrophages can control how a tumour develops. M1 macrophages release proinflammatory cytokines and chemokines that aid in immune surveillance and the promotion of an anticancer immune response. However, M2 macrophages primarily aid in the development of tumours by releasing inhibitory cytokines.

It has been discovered that some proteins can control macrophage polarisation to control the biological activities of ovarian cancer cells. Ovarian cancer patients' ascites contain significant concentrations of Wnt5a, a nonclassical Wnt ligand that encourages the spread of ovarian cancer cells. Wnt5a is the primary regulator of ovarian cancer intraperitoneal metastasis and is mostly expressed by peritoneal mesothelial cells and adipocytes. Wnt5a depletion increased M1 macrophages while decreasing M2 macrophages in a mouse model of ovarian cancer, indicating that Wnt5a fosters an immunosuppressive milieu *via* controlling macrophage polarisation.

The RNA-binding protein SORBS2 can prevent high-grade serous ovarian cancer cells from metastasizing by interfering with M2 macrophage polarisation *via* binding to the 3′-UTR of WFDC1 and IL-17D.

Studies have revealed that tumour cells themselves can affect how macrophages polarise. Additionally, HIF-mediated hypoxia is a critical factor in the development of tumours. Exosomes released by ovarian cancer cells under hypoxic settings, as opposed to those released under normal oxygen conditions, encourage the expression of miR-21-3p, miR-125b-5p, and miR-181d-5p through HIF-1 and HIF-2. Through the SOCS4/5/STAT3 pathway, this further encourages the polarisation of M2 macrophages, which in turn encourages the growth and migration of epithelial ovarian cancer cells. These findings imply that ovarian cancer cells can polarise macrophages by the release of miRNAs and cytokines, which would enhance the development of the tumour.

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