Immunology in gynecological Cancer

The development of cancer may indicate a failure in the immune response through tumor escape mechanisms. The possibility of intervening and intensifying the action of the immune system to produce a beneficial anti-tumor response remains an area of intense research. Tumor cells exhibit a variety of escape mechanisms from immunodetection, leading to an ineffective immune response. In gynecological cancer, several cytokines and cells of the immune system can be used as potential markers, both for the diagnosis and for the prognosis of the disease. The profile of the immune response, whose differentiation and proliferation occurs in the production of certain cytokines, is capable of contributing to the induction of apoptosis, tumor control and regression. Several inflammatory mediators are induced by inflammatory and tumor cells and participate in the formation of cancer, acting as growth or angiogenic factors. One of the escape mechanisms that favors the maintenance of cancer is the triggering of an imbalance in the T helper 1 and T helper 2 response.

The tumor environment in which gynecological cancer develops has been described as a large site rich in pro-inflammatory cytokines and chemokines. Several of these cytokines are produced by the tumor itself, which in addition to stimulating the growth of tumor cells, are also responsible for creating a specific microenvironment that can promote cell proliferation and tumor growth, directly affecting the severity of inflammation.

Another important effector of the immune response to tumors is the peritumoral stroma. This stroma is composed of non-malignant tumor cells, such as cancer-associated fibroblast, mesenchymal cells, innate and adaptive immune cells, vasculature with endothelial cells and pericytes, in addition to an extracellular matrix consisting of structural proteins (collagen and elastin), specialized proteins (fibrillin, fibronectin and elastin) and proteoglycans.

Thus, the immune system plays a complex role in gynecological cancer and can, in different contexts, promote or inhibit tumor growth. The action of the immune system can be different in each gynecological tumor environment: systemic (study of the immune response in the serum of women with cancer), local (study of the tumor microenvironment and peritumoral stroma) and also in the sites around tumors (study of the response immune in the peritoneal fluid). The study of immunology in gynecological cancer is rich and may lead to the discovery of new diagnostic and prognostic markers. Consequently, new therapeutic targets will emerge with these researches, and improve the survival of gynecological cancer in the future.

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