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How does ovarian cancer escape from the host immune system?

Marek Nowak
Polish Mother's Memorial Hospital - Research Institute, Poland

The host response involves both innate and adoptive immune system, which closely cooperate. Generally, the innate immunity is mainly responsible for early detection and elimination of malignant cells, while the adaptive immune system rather controls the tumor progression. However, cancer cells developed variety of strategies to evade the host immune system. They shed surface antigens and down-regulate the expression of molecules necessary for interaction with immune cells. They also produce and release factors (cytokines, enzymes) that exert a modifying effect on the host-adaptive immune response or induce the apoptosis of immune cells. These host-tumor interactions may or may not result in cancer elimination. When the host mediated antitumor immunity is stronger, tumor cells are eliminated; otherwise, cancer cells undergo immune escape and grow rapidly. Emphasizing the dynamic processes between cancer and host immune system, there developed the concept of cancer immunoediting consisting of three phases: elimination, equilibrium, and escape. In the process of elimination nascent transformed cells are recognized and eradicated by innate and adaptive immune system - if all neoplastic cells are eliminated, cancer immunoediting is finished. If all transformed cells are not eliminated at the beginning, immunological pressure leads to the selection of clones with decreased immunogenicity which successively become resistant to the immune system in the equilibrium phase - tumors are usually still not detectable clinically. Developing tumor creates proinflammatory (e.g. IL-6, IL-8) and immunosuppressive (e.g. IL-10, TGF-β) microenvironment leading to the impairment of the host immune function and escape from immunosurveillance resulting in tumor growth and metastases.

mrn@poczta.onet.pl