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The role of the tumor endothelium in leukocyte recruitment in pancreatic cancer.

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Abstract

BACKGROUND: Although pancreatic cancer tissue frequently induces an immune reaction, immunocompetent cells are not able to eliminate the tumor. One potential cause for this ineffective immune response is that a number of active, tumor-cytotoxic T cells are not able to invade into the tumor.

METHODS: A potential barrier for invading leukocytes can be the tumor endothelium, which controls recruitment of leukocytes from circulating blood into the tissue. Although attenuated expression of adhesion molecules on the tumor endothelium has been proposed as a mechanism which suppresses intratumoral leukocyte infiltration, the relevance of adhesion molecules for leukocyte recruitment in tumor tissue is poorly understood.

RESULTS: The leukocyte extravasation in normal pancreas during acute pancreatitis follows the "classic" leukocyte recruitment cascade and is controlled by the overexpression of endothelial adhesion molecules, such as selectins, intracellular adhesion molecule-1, and platelet endothelial cell adhesion molecule-1. In contrast to acute inflammation in normal pancreas, leukocyte recruitment in pancreatic cancer is a slow process, which does not show a strong dependence on intracellular adhesion molecule-1. In addition, pancreatic cancer has a high degree of heterogeneity of both immunogenic properties and the distribution of tumor-infiltrating leukocytes, such as CD8(+), CD4(+), or regulatory T cells.

CONCLUSION: Additional studies may clarify whether T cell recruitment and their activity in pancreatic cancer can be enhanced by modulation of endothelial adhesion molecules.

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