The Molecular Biology of Peritoneal Carcinomatosis from Gastrointestinal Cancer
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Abstract

Introduction: Peritoneal carcinomatosis is a frequent form of disease progression in gastrointestinal cancer, and all too often is a preterminal event with a median survival of only 6 months. Despite the introduction of aggressive surgical and chemotherapeutic approaches, any significant improvement in survival is unlikely until we better understand the molecular biology of peritoneal metastasis.

Methods: A Medline search and review of references was undertaken to identify all manuscripts in the English language concerned with peritoneal metastasis from gastrointestinal cancer.

Results: Peritoneal carcinomatosis involves a complex sequence of interdependent steps. The injured peritoneum is a rich source of cytokines and growth factors that facilitate tumour proliferation and invasion in the postoperative abdomen. Peritoneal tumour adhesion is dependent on adhesion molecules, such as CD44, and the β-1 integrins. Invasion of the mesothelium involves, at least in part, a process of tumour-induced mesothelial apoptosis. Matrix metalloproteinases, such MMP-7, facilitate stromal invasion, but the role of other proteases in invasion remains to be elucidated. To date, the significance of angiogenesis in the peritoneal metastatic cascade is unknown.

Conclusion: The molecular biology of peritoneal carcinomatosis is only just beginning to be understood. Further research into the mediators of the peritoneal metastatic cascade is needed if more effective therapeutic strategies are to be developed for this invariably fatal, yet unfortunately common, condition.

Key words: Gastrointestinal neoplasms, Neoplasm metastasis, Peritoneum

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