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Understanding the Mechanisms of Breast Cancer Metastasis: Theoretical Considerations and Clinical Implications

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Commentary

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DESCRIPTION

The permeation theory of breast cancer spread was the stimulus to the development of increasingly radical surgery. This theory was the first casualty of a greater understanding of the biology of breast cancer. In 1931 Gray demonstrated that the lymphatics around a primary breast tumour were neither obliterated nor filled with cancer cells, even when axillary nodes were involved by tumour. This weakened the en bloc principle of radical surgery and contributed to the rationale for the less disfiguring modified radical mastectomy in which the breast is removed together with the axillary contents, whilst preserving the pec-toralis major muscle. The great proponents of this operation in the 1930s were Patey in England and subsequently Auchincloss and Madden in the United States. In 1955, Engell demonstrated venous dissemination of breast cancer cells from early operable tumours. This also dealt a blow to the permeation theory which stated that haemotogenous spread of tumours occurred only very late in the pathophysiology of breast cancer. Subsequently, Fisher and Fisher's work demonstrated that lymph nodes were poor barriers to the spread of cancer cells. In a classic series of experiments in rabbits, they demonstrated that tumour cells could pass easily through lymph nodes into efferent lymphatics and also into veins through lymphaticovenous communications.

The above observations helped to foster towards the theory of breast cancer spread which in turn led to a new era in the surgery of breast cancer. It became apparent that radical surgery had reached its anatomical limits without contributing to a reduction in mortality from breast cancer. This was because the concept of local origin provides a basis for cure only if the diagnosis can be made before dissemination has taken place. The stage at which an occult or even symptomatic neoplasm disseminates is extremely variable and is dependent on many factors. One factor long thought to enhance tumour cell dissemination is the effect of handling the tumour during surgery. Trauma to tumours increases both cell shedding and metastasis in animal models. Early studies of tumour cell shedding in humans were beset by problems with sampling and cell identification and this led to a decline in interest in the subject. Recently, there has been renewed interest in this proposed mechanism of tumour cell dissemination. This is because of an enhanced ability to detect more reliably small numbers of carcinoma cells among large numbers of haematopoietic cells using monoclonal antibodies against epithelial-restricted epitopes or using quantitative polymerase chain reaction. A recent study using very sensitive immunohistochemical techniques on selective venous samples before, during and after breast cancer surgery has demonstrated increased shedding of breast cancer cells into the circulation during surgery. Furthermore, the likelihood of cell shedding was directly related to tumour angiogenesis as measured by vascular density of the tumor.

However, tumour angiogenesis is probably not the only mechanism involved in the ability of a tumour to metastasize. Transformed cells also require a reduction in adhesiveness to detach them and enter the circulation. Thus, for migration to occur, the affinity between cancer cells and endothelium or lymphatic channels need to change. For a cancer cells to attach to a particular target organ, further changes in expression of adhesion receptor in the invading cell and the target tissue are necessary.