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An Short Overview of Breast Cancer and Its Management

Ben Franklyn*

Department of Oncology, Ege University Faculty of Medicine, Bornova, Turkey

Description

Invasive breast cancer is the ultimate result of a complex evolutionary process that began in the TDLU of the mammary gland. It is a multi-step pathway involving cell proliferation, differentiation, aberrant intracellular expression, and signaling that leads to an imbalance between mammary epithelial cells and the breast tissue microenvironment. In the last phases, disturbance of epithelial-stromal balance can result in epithelial carcinogenesis, increasing stromal invasion, and metastatic dissemination. Although we are aware of some of the histopathologic aspects in these processes, the molecular profile of these events is still unknown and remains a topic for future research. The current knowledge indicates that the dual functional activity of the breast tissue microenvironment on breast carcinogenesis.

It can encourage it at times, but it also has the ability to inhibit malignant transformation and remove cells with a malignant phenotype. The earliest publications indicating the active involvement of tissue microenvironment signaling activity on breast carcinogenesis and the nature of malignant cell clones, such as tumour grade, date back more than 30 years. Over the last decade, technological advances in molecular and tissue cell biology, primarily through gene expression profiling approaches, have enabled us to detect and actively describe the role of myoepithelial cells, fibroblasts, adipocytes, immune system cells, growth and hormone factors, extra-cellular matrix, and epithelial-mesenchymal transition in the complicated process of progressive epithelial cell transformation into pre-invasive and invasive forms of breast cancer. These "structures" of breast tissue microenvironment are now regularly defined by mammary pathologists when reporting results from breast lesion biopsies.

Furthermore, these data demonstrate cancer phenotype-specific links in morphologic variability or attainment of invasive state. The dual form (barrier escape/failure) of DCIS transition to invasive breast cancer is a typical example of such biologic heterogeneity in microenvironment influenced and regulated carcinogenesis. The subject rupture of myoepithelial cells line and basement membrane tumour cells is related with barrier escape, allowing them to move and disseminate into surrounding stroma. However, when the barrier fails, the myoepithelial cell line and basement membrane are disrupted to varying degrees, followed by significant lymphocyte infiltration and myofibroblast accumulation. Aside from that, evidence in the literature show that microenvironment signaling has a direct influence on cancer biologic traits and later clinical behaviour.

Finally, the discovery and presentation of the "premetastatic niche concept" is an narrative highlighting the critical function of the breast microenvironment in the progression of mammary carcinomas. This is the concept that describes the active signaling role of the microenvironment on progenitor cells, stem cells, or aggressive cancer clonal cells in the role of migration, malign transformation, and location of future metastases, in conjunction with active host cell participation, primarily from the host bone-marrow cell migration able population. The active role of microenvironment components in the process of breast tumorigenesis and selection of targeted therapy in patients resistant to endocrine treatment is discussed in this special issue at the level of activity of cytotoxic suppressor cells, neoangiogenic, immunosuppressive factors, and role of adipocytes in the mammary tissue microenvironment.

The advancement of health-directed technology is also linked to advancements in genetic profiling of breast carcinomas and the following emergence of customized therapy. Breast carcinoma is widely recognized as one of the most varied kinds of human cancer, including a diverse spectrum of epithelial and stromal components with significant functional influence on the afflicted patient. Oncologic practice has demonstrated that stratifying patients with breast cancer into risk groups and subsequent treatment protocols based solely on common prognostic and predictive parameters is ineffective for everyone and has limitations in predicting future biologic behaviour of the disease.

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***Address for Correspondence:** *Dr. Ben Franklyn, Department of Oncology, Department of Oncology, Ege Aceniversity Medical Faculty, Bornova, Turkey; E-mail: Benyn@gmail.com*

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