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Histologic Changes and Clinical Implications in Breast Cancer

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Introduction

Neoadjuvant therapy (cytotoxic or endocrine therapy before surgery) for breast cancer has become the norm, providing the chance to gauge and quantify response in the resection specimen. Important prognostic information that could direct subsequent therapy is provided by correlation with radiography, cassette mapping, and histologic examination with a semi-quantitative reporting system like residual cancer burden (RCB). Histologically, the tumour bed should be recognised as a collagenized zone lacking normal breast epithelium and exhibiting enhanced vasculature. Because leftover tumour cells may be tiny and dyscohesive and have hyperchromatic small, big, or numerous nuclei with clear, foamy, or eosinophilic cytoplasm, identifying treated residual carcinoma may require meticulous high power inspection [1].

Description

Adenocarcinomas, which make up more than 95% of breast cancers, are the most common breast malignancies. 4 The most typical type of invasive breast cancer is invasive ductal carcinoma (IDC). It is responsible for 55% of breast cancer cases at the time of diagnosis. 5 Breast cancers develop from the same terminal duct lobular unit segment (TDLU). Invasive breast cancer and its histological variations have established typing. Ductal carcinoma in situ (DCIS) and IDC are the two main subtypes of breast cancer. DCIS is a duct- and lobule-confined intraductal growth of epithelial cells that is noninvasive and potentially cancerous. Carcinoma that has invaded or infiltrated the stroma through the duct wall and is malignantly abnormally proliferating in the breast tissue is referred to as an invasive or infiltrative carcinoma [2].

It is a neoplastic proliferation of epithelial cells that is only found in the ducts or lobules and is characterised by nuclear and cellular atypia, potential malignancy, and both obligate and nonobligate inclinations to progress to invasive breast cancer. 1 The outer ductal layer's myoepithelial cells are often still there, however they could be weaker or fewer in quantity. Lobular cancerization is the term used to describe how DCIS spreads through the ducts and into the lobular acini, resulting in large lesions [3]. DCIS is thought to be a precursor lesion for the later onset of invasive carcinoma with a higher risk index factor than would be anticipated in women without DCIS. With the widespread use of screening mammography and rising breast cancer awareness in the United States since 1983, a significant rise in the detection of these lesions has been made.

Breast cancer is a very variable condition that varies significantly between people (intertumor heterogeneity) and even within each individual tumour (intratumor heterogeneity). The staging systems and histopathologic division of breast cancer reflect the clinical and morphologic intertumor heterogeneity. The foundation for targeted therapy is heterogeneity in the expression of recognised prognostic and predictive biomarkers, hormone receptors, and human epidermal growth factor receptor 2 oncoprotein. Multigene tests can investigate the genetic heterogeneity of tumours and improve stratification into low- and high-risk groups for individualised therapy. Molecular classifications are indications of genetic tumour heterogeneity. Intratumor heterogeneity presents diagnostic and therapeutic problems since it occurs at the morphologic, genomic, transcriptomic, and proteomic levels.

Determining the tumours' potential for metastasis informs therapy choices in breast cancer care. It is common practise to group tumours into subgroups that are prognostic of outcome using clinical factors that represent metastatic potential (e.g., lymph node status, tumour size, histologic grade), or forecast for endocrine response (e.g., oestrogen and progesterone receptors). These factors, however, are unable to accurately identify which individuals would respond well to existing treatment methods, benefit from adjuvant treatment, or perform badly without it. Despite morphological homogeneity, several tumour subgroups are linked to significant clinical variability, which confuses their clinical value. Such clinical variability may be resolvable at the genetic level, according to recent investigations employing DNA microarrays.

One of the characteristics of malignancy is tumour heterogeneity. Different people's breast carcinomas exhibit intertumor heterogeneity. Intratumor heterogeneity results from the presence of several cell types within a single tumour. Early studies used the detection of intratumor cell populations with various properties, such as tumorigenicity, treatment resistance, and metastatic potential, to characterise tumour heterogeneity. Although the heterogeneity of breast cancer at the cellular level was recognised as early as the eighteenth century, oestrogen receptor (ER) testing, which was developed about 30 years ago, was the first to demonstrate its therapeutic importance (6). Differentiations in clinical behaviour and treatment response were assumed to be caused by variations in ER expression between tumour types or between various cell populations within a single tumour.

The grade of breast cancer also emphasises the heterogeneity of the tumour. The percentage of the tumour that is organised into glands and tubular structures, the level of nuclear pleomorphism, and the mitotic rate are the three morphologic characteristics that are evaluated to determine the grade (low, middle, high). A strong prognostic indicator, the grade of breast cancer is taken into account by clinical decision-making tools like the Nottingham Prognostic Index and Adjuvant. By using proteomic, genomic, and transcriptome studies, different grades of breast cancer also exhibit diverse profiles. For ER-positive tumours, grade continues to be an independent predictive predictor in multivariate models with gene signatures [4,5].

Conclusion

Often referred to as "triple-negative" breast carcinomas, breast cancers that lack the ER, PR, and HER2 proteins exhibit a wide range of histological, genetic, prognostic, and therapeutic response characteristics. According to recent research, triple-negative (ER-, PR-, and HER2-) breast cancers can exhibit nuclear expression of the androgen receptor (AR) in 12–55% of cases. AR expression's prognostic value in triple-negative carcinomas is debatable, but it is connected to better survival in other tumour subtypes. Current clinical trials examining AR antagonists (such as bicalutamide and enzalutamide) in triple-negative breast carcinomas that are AR+ (defined as nuclear staining in 10% of tumour cells by IHC) show encouraging outcomes.

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Conflict of Interest

There are no conflicts of interest by author.

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