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Opinion Article

Breast Cancer Prognostic Markers: Are They Really Addressing the Issues?

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DESCRIPTION

Breast cancer is a contrasting disease with an asymmetric morphology, molecular features, which makes it atypical in response to therapies. Conventionally, prognostic markers in breast cancer is based on the clinico-pathological parameters and detached molecular markers, but on the other hand breast cancer prognosis behaves to some extent asymmetrically in different ethnic groups; although this is a debateable topic, but this situation still exists.

Family history does play a different role in prognosis according to a contemporarily published study where an accumulated number of ER negative &PR negative breast cancer was acclaimed among younger Spanish women who have a family account of the disease. The established/routinely used prognostic markers which are being used by some of the highly respected institutes are ER, PR, Her-2, p53, CD31, Ki-67/PCNA. Trastuzumab is being offered to Her-2 positive patients who will benefit with this monoclonal drug or in other words Her-2 is being performed on a selective group of patients. Whereas ER, PR, p53, CD31, Ki-67/PCNA are being performed on almost every breast cancer patient.

Histologically, the majority of breast cancers (65% to 80%) belong to an ascetic subtype, invasive ductal carcinoma. This communicatively contours the use of type as a prognostic mediator Therefore, assessment of tumor behavior for any breast cancer case has been based on deltoid parameters: tumor size, lymph node condition, and histological grade. Tumor size is a good prognostic marker for metastasis or otherwise in lymph node negative patients, although patients with small tumors (<1 cm) after the surgical removal are not offered any further treatment, have algorithmically a 12% adventitious of periodicity. Lymph node status is still appraised the best prognostic indicator of relapse, but with the commencement of the drugs like tamoxifen and trastuzumab, a more molecular characterization of the tumour is in great demand.

There are about 54 drugs which have been approved by the Food and Drug Administration for treatment; so the oncologist thus requires some kind of may be accurate advocacy about the molecular characteristic of the tumour to deal with some precision. More recently, a battery of four prognostic markers (ER, PR, HER2 and Ki67) has been shown to have a high prognostic impact which could be similar to that of gene expression assays. Some other markers, like serine protease urokinase-type plasminogen activator (uPA) and its inhibitor (plasminogen activator inhibitor type-1;PAI-1) have reached the evidence level by where it can be judged by the American Society for Clinical Oncology as acceptable for clinical use in patients with newly diagnosed node negative breast cancer using an ELISA assay.

Gene assertion profiling assays have assorted breast cancer into five molecular subtypes; luminal A, luminal B, HER2, basal-like, and normal-like. Luminal A (ER+ and Ki67 low) cancers are appeared to adhere the eclipse prognosis; HER2 and basal cancers (also sometimes accredited to as triple negative tumors) have the worst prognosis, and the prognosis for luminal B (ER+ and Ki67 high) cancers is in between.

Recently, protein biomarkers appraised by reverse phase protein arrays show consequential intra-tumour heterogeneity in breast cancer, and 15 additional proteins have been assayed belonging either to the identical protein family as claimant proteins or involved in downstream signaling of the candidate molecules. In another current study, it was acclaimed that many of these proteins are complementary with uPA and PAI-1 assertion in primary breast cancers and might be dictatorial for uPA and PAI-1 accompanied tumor augmentation and metastasis. The expression of uPA was correlated with expression of ER and the Stat3/ERK pathway while PAI-1 was affiliated with Akt signaling and regulation of the HER family. As the activated proteins, the phosphorylated proteins that are frequent appraised include pAkt, p1086EGFR, p1148EGFR, pER, pERK, pGSK3b, pHer2, pHer3, pPDGFR, pp38, pPR, pPTEN, p727STAT3 and p705STAT3."

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