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Editorial

From Immunohistochemistry to Molecular Testing for the Precision Cancer Management of Breast Cancer

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Introduction

Breast cancer is not a single disease. It is a biologically heterogeneous collection of malignancies, each with its own molecular fingerprint, natural history, and therapeutic vulnerability. For decades, the clinical management of breast cancer rested on a triad of morphology, staging, and the immunohistochemical (IHC) expression of three cardinal biomarkers: estrogen receptor (ER), progesterone receptor (PR), and human epidermal growth factor receptor 2 (HER2), supplemented by the proliferation index Ki-67.[1] This IHC-based surrogate classification formalised by the St. Gallen International Expert Consensus has undeniably transformed breast oncology, stratifying patients into five clinically actionable subtypes and guiding systemic therapy decisions with meaningful prognostic discrimination.[2] Yet the limitations of this approach have grown increasingly apparent as precision oncology matures: IHC surrogates achieve molecular concordance of only 80–85% with gene expression profiling, are subject to inter observer variability, and critically fail to capture the dynamic intra-tumoral genomic complexity that underlies treatment resistance.[3]

The clinical imperative to move beyond IHC is now well evidenced. Multigene expression assays Oncotype DX, MammaPrint, Prosigna, and Endo Predict have demonstrated robust prospective validation as tools for chemotherapy decision-making in hormone receptor-positive, HER2 negative early breast cancer, with the TAILORx and MINDACT trials establishing their capacity to spare a substantial proportion of patients from unnecessary cytotoxic therapy.[4,5] In the HER2 positive space, the DESTINY Breast04 trial redefined the therapeutic boundary of HER2 classification, demonstrating that trastuzumab-deruxtecan (T-DXd) achieves significant survival benefit in HER2 low tumours- IHC score 1+ or 2+ with negative in situ hybridization a category entirely invisible to conventional HER2-positive/negative binary classification.[6] This landmark finding alone renders the binary IHC framework clinically obsolete for a subset of patients and underscores the need for refined biomarker stratification.

For triple-negative breast cancer (TNBC), historically defined by the absence of ER, PR, and HER2 expression, the paradigm shift has been equally dramatic. Next-generation sequencing (NGS) based profiling has dissected TNBC into at least six molecular subtypes including basal like 1 and 2, immunomodulatory, mesenchymal, mesenchymal stem like, and luminal androgen receptor subtypes each with distinct therapeutic vulnerabilities.[7] BRCA1/2 germline mutation testing now guides PARP inhibitor use, PD-L1 IHC combined with immune gene signatures directs pembrolizumab eligibility, and PIK3CA/AKT1 pathway mutations are emerging targets in advanced disease.[8,9] IHC alone cannot adequately navigate this complexity.

Against this evolving landscape, the article by Rahman et al. published in this issue presents a timely and clinically significant dataset. The authors characterise the IHC-based molecular subtype distribution of 107 invasive breast carcinoma patients from a Bangladeshi tertiary centre, revealing that TNBC constitutes 30.8% of cases substantially exceeding global rates of 15–20% while Luminal A accounts for only 26.2%.[10] These findings delineate a population with a

disproportionate burden of the most therapeutically challenging subtype, in a setting where NGS based molecular testing remains largely inaccessible. Significant associations between molecular subtypes and histological grade ($p<0.004$) and lymph node metastasis ($p<0.035$) validate the biological credibility of their IHC classification and highlight the indispensability of even basic molecular stratification in guiding oncological decisions.

The study from Rahman et al. is therefore both an endorsement of IHC as the essential foundation in resource limited settings and an implicit call to action for the next step. IHC remains irreplaceable as the accessible, affordable, and reproducible cornerstone of breast cancer diagnostics globally; however, it must increasingly be regarded as the beginning, not the end, of the molecular diagnostic pathway.[11] As NGS platforms become more affordable and liquid biopsy technologies mature, integration of comprehensive genomic profiling into routine breast oncology workflows is no longer a futuristic aspiration but an immediate translational priority even in low and middle income countries.[12]

The trajectory is clear: precision breast cancer management demands a staged, scalable molecular diagnostic framework in which IHC provides the initial subtype scaffold, and targeted molecular testing multigene assays, hotspot mutation panels, and ultimately comprehensive NGS refines therapeutic decision making at each clinical decision point. Bridging this translational gap equitably, across all geographies and economic contexts, remains the defining challenge of the next decade in breast oncology.

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