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Opinion Article

Metaplastic Breast Cancer's Therapeutic Environment

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DESCRIPTION

There is a significant unmet demand for Metaplastic Breast Carcinomas (MPBC), which are uncommon, aggressive, and often chemorefractory tumours. Even though the majority are "triple negative" and do not express ER, PR, or HER2 receptors, MPBC have poorer prognoses than typical triple negative invasive tumours. The most common somatic mutations in TP53, PIK3CA, and PTEN are found in MPBCs, which are genetically diverse and may be amenable to innovative targeted therapeutics, according to recent research. Additionally, these tumours have been linked to tumor-infiltrating lymphocytes and overexpressed PD-L1, which indicates an endogenous immune response and supports the use of immunotherapies as a form of therapy. This study concentrates on treatment possibilities for this challenging subtype of breast cancer, and doctors should take targeted medicines and immunotherapies into account as a part of on-going clinical studies.

As 0.2–5% of all breast cancers, Metaplastic Breast Carcinoma (MPBC) is often exceedingly aggressive and has worse clinical outcomes than Triple-Negative Invasive Breast Cancer (TNBC). The most common histological subtype is squamous cell carcinoma, and it is made up of ductal, squamous and chondroid, and spindle components. Low-grade adenosquamous carcinoma, fibromatosis-like metaplastic carcinoma, squamous cell carcinoma, spindle cell carcinoma, metaplastic carcinoma with mesenchymal differentiation, mixed metaplastic carcinoma, and myoepithelial carcinoma are additional subgroups that the World Health Organisation (WHO) has identified. The majority of MPBC cases appear as a rapidly expanding breast mass, which accounts for the higher size of MPBC at presentation compared to Invasive Ductal Carcinoma (IDC) in women over the age of 50. These tumours can be found using normal mammography in combination with ultrasonography because they present on imaging as ill-defined masses without distinct radiological characteristics. A localized illness will be present in nearly 90% of individuals with MPBC, but 50% of these patients will later develop distant metastasis, a risk that is twice as high as that of TNBC. *de novo* metastatic disease is observed in the remaining 10% of MPBC patients. Axillary lymph node metastasis is less common than metastases to other forms of breast cancer, occurring in only 15–25% of surgical cases. When compared to TNBC, MPBC substantially more frequently develops lung-only metastases (42 *vs.* 18% of metastatic patients). With an average Overall Survival (OS) of less than one year in the metastatic setting—as low as 3.4 months in one study—MPBC frequently has poor outcomes that are worse than TNBC. In comparison to 73% for TNBC and 89% for general IDC, the 5-year OS varies from 54% to 69%. Tumour size more than 5 cm, lymph node involvement, and elevated Ki67 are characteristics that indicate poorer 5-year OS and Progression-Free Survival (PFS). The aggressive clinical behaviour of MPBC is a result of EMT, which encourages metastasis growth by facilitating invasion and migration. For instance, it was discovered that MPBC had low levels of expression of EP300, a transcriptional activator of E-cadherin, which resulted in a more malignant phenotype and the development of treatment resistance. Similar to this, a retrospective examination of 27 original MPBC tumours revealed that ZEB1, an inducer of EMT, was upregulated and E-cadherin expression was decreased or missing in all non-glandular metaplastic regions.

Demonstrating in-person proof that non-glandular MPBC include EMT inducers. This "EMT signature" is associated with a decreased chance of survival and a lack of a Pathologic Complete Response (pCR) following neoadjuvant chemotherapy. Studies have linked MPBC's chemotherapy resistance to stem cell-like characteristics, as evidenced by the non-glandular components' elevation of the tumour stem cell marker protein Aldehyde Dehydrogenase-1 (ALDH-1) and CD44/CD24 ratios.

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