Immunohistochemical Expression of Galectin-3 and MUC1 in Tumour Cells and Tumour-Associated Stromal Cells in Invasive Breast Carcinoma: Association with Clinicopathological Parameters

Dr. Dommati Mounika *
Final-Year Postgraduate Resident, Department of Pathology and Laboratory Medicine, All India Institute of Medical Sciences (AIIMS), Raipur, Chhattisgarh, India

Dr. Yashita Gupta
Associate Professor, Department of Pathology and Laboratory Medicine, All India Institute of Medical Sciences (AIIMS), Raipur, Chhattisgarh, India

Dr. Rakesh Kumar Gupta
Additional Professor, Department of Pathology and Laboratory Medicine, All India Institute of Medical Sciences (AIIMS), Raipur, Chhattisgarh, India

Dr. Radhakrishna Ramchandani
Associate Professor, Department of General Surgery, All India Institute of Medical Sciences (AIIMS), Raipur, Chhattisgarh, India

Dr. Amit Kumar
Assistant Professor, Department of Medical Oncology, All India Institute of Medical Sciences (AIIMS), Raipur, Chhattisgarh, India

Abstract

Background: Breast carcinoma progression is increasingly understood as tumour–stroma crosstalk rather than epithelial changes alone. Galectin-3 and MUC-1 regulate adhesion, invasion and metastasis, but their expression in cancer-associated fibroblasts (CAFs) remains underexplored.

Aim: To evaluate Galectin-3 and MUC-1 expression in tumour-cells and CAFs by immunohistochemistry and correlate findings with clinicopathological parameters.

Methods: Ambispective study included 100 cases of invasive breast carcinoma. Galectin-3 and MUC-1 were assessed using H-score and Immunoreactive-score, respectively, in tumour-cells and CAFs. Associations were analysed using chi-square, Mann–Whitney/Kruskal–Wallis, Spearman correlation and multivariable logistic regression.

Results: Tumour-cell Galectin-3 negativity increased with nodal burden (27.1% in pN0 vs 85.7% in pN2–3; $p<0.001$) and was associated with LVI ($p=0.017$). Conversely, high CAF Galectin-3 increased with nodal burden, LVI and PNI (all $p<0.05$), demonstrating an inverse tumour–stromal relationship. Tumour-cell MUC-1 expression showed limited clinicopathological associations; however, MUC-1 heterogeneity increased with nodal progression, from 20.8% in pN0 to 55.6% in pN1 and 76.2% in pN2–3 ($p<0.001$). Heterogeneity was associated with tumour-cell Galectin-3 ($p=0.024$) and CAF Galectin-3 ($p=0.001$). Low/negative tumour-cell Galectin-3 and high CAF Galectin-3 were associated with heterogeneous MUC-1 staining. An integrated biomarker score showed a graded association with nodal burden ($p<0.001$). Multivariable analysis identified low/negative tumour-cell Galectin-3, high CAF Galectin-3, MUC-1 heterogeneity and LVI as independent predictors of nodal metastasis.

Conclusion: The significance of Galectin-3 and MUC-1 depends on cellular compartment, spatial distribution and heterogeneity rather than expression quantity alone. Compartment-based IHC assessment may provide a foundation for integrated prognostic biomarkers in practice.

Keywords

Invasive breast carcinoma, galectin-3, mucin-1, cancer-associated fibroblasts, tumour–stroma interaction, immunohistochemistry, nodal metastasis, lymphovascular invasion.